From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
To: Amy Kostant <amy@sciencecom.org>
Sent: 1/22/2015 1:57:58 PM
Subject: Re: stories

Wow, cool, the Daily Mail!

From: Amy Kostant <amy@sciencecom.org>
Date: Thursday, January 22, 2015 1:48 PM
To: patricia hunt <pathunt@vetmed.wsu.edu>
Cc: Emily Copeland <emily@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Subject: RE: stories

Thank YOU for such your excellent work!
Check out this one: http://www.dailymail.co.uk/health/article-2921840/Why-sperm-counts-falling-Contraceptives-drinking-water-chemicals-plastics.html

---

From: Hunt, Pat [mailto:pathunt@vetmed.wsu.edu]
Sent: Thursday, January 22, 2015 3:05 PM
To: Amy Kostant
Subject: stories

Hi Amy-

I thought Brian did a really nice job and so did Lynne Peeples (http://www.huffingtonpost.com/2015/01/22/chemicals-infertility-endocrine-disruptors_n_6524536.html). I spent a lot of time on the phone with Doug Main this morning and he seems quite keen on it.

Thanks for all of your help!

Pat

From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
To: Pete Myers <jpmyers@ehsic.org>
Sent: 7/25/2017 8:54:38 AM
Subject: Re: talk this afternoon?

It was interesting.

My window of opportunity is narrow - could you talk between 2 and 3?

Sent from my iPhone

> On Jul 25, 2017, at 11:22 AM, Pete Myers <jpmyers@ehsic.org> wrote:
>
> How did your day go yesterday?
>

From: <JPMyers@ehsic.org>
To: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Sent: 7/25/2017 9:12:05 AM
Subject: Re: talk this afternoon?

"It was interesting." mmm.... doesn't sound good.

How about we zoom at 2. If you can't use video, call in on the dial in number.

Topic: Pat and Pete
Time: Jul 25, 2017 2:00 PM Eastern Time (US and Canada)

Join from PC, Mac, Linux, iOS or Android: https://zoom.us/j/[redacted]

Or iPhone one-tap (US Toll): +16465588656,,[redacted] or +14086380968,,[redacted]

Or Telephone:
  Dial: +1 646 558 8656 (US Toll) or +1 408 638 0968 (US Toll)

-----"Hunt, Pat" <pathunt@vetmed.wsu.edu> wrote: -----

To: Pete Myers <jpmyers@ehsic.org>
From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Date: 07/25/2017 11:54AM
Subject: Re: talk this afternoon?

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Sent from my iPhone

> On Jul 25, 2017, at 11:22 AM, Pete Myers <jpmyers@ehsic.org> wrote:
>
> How did your day go yesterday?
>

From: Patricia Hunt <pathunt@vetmed.wsu.edu>
To: Amy Kostant <amy@sciencecom.org>
Sent: 1/2/2014 9:17:36 AM
Subject: Re: talk with reporters?

Hi Amy-

Happy New Year! Yes, I had a lovely holiday. We spent two weeks in Hawaii and returned the day before Christmas and spent Christmas week with my dad. It was lovely to take such a long break from work and I feel refreshed and ready to get back in the saddle.

I would, of course, be willing to read Gail's paper and talk with reporters if the need arises. Can you send me the pdf?

I will be in DC Feb 11-12. I haven't booked tickets yet but, if you are around, I will try to book a late departure and hope we can have coffee together. I'll be in touch.

Pat

From: Amy Kostant <amy@sciencecom.org>
Date: Thursday, January 2, 2014 7:15 AM
To: "pathunt@wsu.edu" <pathunt@wsu.edu>
Cc: Emily Copeland <emily@sciencecom.org>
Subject: talk with reporters?

Hi Pat
I hope youve had a lovely holiday.
Gail Prins has a paper on BPA and prostate cancer publishing next week in *Endocrinology*. Would you be willing to read the paper and take a call or two if we get reporters who ask to talk with experts other than the author? It would mean being generally available between today and next Tuesday.
I think you know this drill -- we rarely get these requests, but when we do its incredibly helpful to have someone up to speed and available. There wouldnt be more than one or two requests at the very most. Thanks for considering.
All best wishes for a joyful and healthy new year.
And when are you planning to be in DC? I would love to get together.
Amy

****
Amy Kostant
Science Communication Network
Office: 301-654-6665
Cell: 202-255-6665
amy@sciencecom.org

From: Pete Myers <jpmyers@ehsic.org>
To: "R. Thomas Zoeller" <tzoeller@bio.umass.edu>
CC: Laura Vandenberg <lvandenberg@schoolph.umass.edu>, Shanna Swan <shanna.swan@mssm.edu>, Harvey Karp <dr.karp@thehappiestbaby.com>, "Prins, Gail" <gprins@uic.edu>, Bruce Lanphear <blanphear@sfu.ca>, Joan M Cranmer <cranmerJoanM@uams.edu>, "Cory-Slechta, Deborah" <deborah_cory-slechta@urmc.rochester.edu>, Peter Orris <porris@uic.edu>, "Prof. Fred vom Saal" <vomsaalf@missouri.edu>, Terry Collins <tc1u@andrew.cmu.edu>, Howard Snyder <snyderh@email.chop.edu>, Peter DeFur <environsc@gmail.com>, Shuk-Mei Ho <shuk-mei.ho@uc.edu>, Ted Schettler <tschettler@igc.org>, "Ozonoff, David" <dozonoff@bu.edu>, Tyrone Hayes <tyrone@berkeley.edu>, Tracey Woodruff <WoodruffT@obgyn.ucsf.edu>, "Dr. Steve Heilig" <heilig@sfms.org>, "Stahlhut, Richard" <richard_stahlhut@urmc.rochester.edu>, Sheldon Krimsky <sheldon.krimsky@tufts.edu>, "Landrigan, Philip" <philip.landrigan@mssm.edu>, Pat Hunt <pathunt@wsu.edu>, Andreas Kortenkamp <andreas.kortenkamp@brunel.ac.uk>, Russ Hauser <rhauser@hohp.harvard.edu>, Bruce Blumberg <blumberg@uci.edu>, Amy Kostant <amy@sciencecom.org>, "Bernard Weiss" <Bernard_Weiss@urmc.rochester.edu>, Kalee Kreider <kaleekreider@gmail.com>, Emily Copeland <emily@sciencecom.org>, "Carl-Gustaf Bornehag" <caguborn@kau.se>, Michael Antoniou <michael.antoniou@kcl.ac.uk>, Steve Gilbert <sgilbert@innd.org>, Leonardo Trasande <leonardo.trasande@nyu.edu>, Amy Itescu <itescua@UCMAIL.UC.EDU>, Joseph Allen <jgallen@hsph.harvard.edu>
Sent: 12/13/2016 10:17:36 AM
Subject: Re: The Shredding of Exponent

Amy would it be worth Tom sending a letter to the editor of FairWarning about this?

> On Dec 13, 2016, at 1:10 PM, R. Thomas Zoeller <tzoeller@bio.umass.edu> wrote:
>
> Hi All I have to admit that the story about EDCs did give some slightly false impressions. Specifically, when Lamb et al came out with their scathing review of the UNEP/WHO State of the Science report, we decided not to bother with a response, because their arguments were just silly and transparent and because the kerfuffle over the Dietrich open letter and the following response made it seem like a debate. But when Crop Life International had a side event at ICCM3 focusing on the lamb critique, we decided that we should respond. The response was published in the same journal (see below) and I think this response is an excellent dissection of what industry does. So, while the industry responded to my statement like school children taunting it takes one to know one, the basis for my statement was much more thorough and scholarly and was not so easily dismissed.
>
> If the CEO of Exxon Mobile really becomes the US Secretary of State, it will be a transition in the debate about running the US government like a business to running the US government AS a business for the personal gain of a few. Of course, this isnt far from what is happening already...
>
> <Bergman et al Reg Tox Pharm 2015.pdf>

R. Thomas Zoeller, Professor
Biology Department
University of Massachusetts Amherst
611 N Pleasant St.
Amherst, MA 01003

ph: (413) 545-2088
Fax: (413) 545-3243

http://www.bio.umass.edu/biology/about/directories/faculty/r-thomas-zoeller

On Dec 13, 2016, at 12:52 PM, Laura Vandenberg <lvandenberg@schoolph.umass.edu> wrote:

Agreed - this is a great piece that deserves some web traffic.

I was recently introduced to the concept of 'gas-lighting' mostly in the context of the president-elect, who uses it to suggest that questioning his knowledge, abilities, preparation or experience is just more evidence of a biased media. This is true for Exponent's response to Tom Z's comments about manufactured doubt for EDCs... raising this concern means there really isn't any science to defend. This is frustrating, mainly because it is quite effective. I wonder if any science communication folks have thought of strategies to combat these approaches...

Best,
Laura

Laura N. Vandenberg, PhD
Assistant Professor
University of Massachusetts Amherst
School of Public Health & Health Sciences
Department of Environmental Health Science
686 N. Pleasant Street
171A Goessmann
Amherst, MA 01003
Tel: 413.577.7405
lvandenberg@schoolph.umass.edu

From: Swan, Shanna <shanna.swan@mssm.edu>
Sent: Tuesday, December 13, 2016 10:32 AM
To: Pete Myers
Cc: Karp, Harvey; Prins, Gail; Bruce Lanphear; Cranmer, Joan; Cory-Slechta, Deborah; Peter

Orris; Fred vom Saal; Terry Collins; Howard Snyder; Peter DeFur; Shuk-Mei Ho; Tom Zoeller; Ted Schettler; Ozonoff, David; Tyrone Hayes; Tracey Woodruff; Dr. Steve Heilig; Stahlhut, Richard; Sheldon Krimsky; Landrigan, Philip; Pat Hunt; Andreas Kortenkamp; Russ Hauser; Bruce Blumberg; Amy Kostant; Bernard Weiss; Kalee Kreider; Laura Vandenberg; Emily Copeland; Carl-Gustaf Bornehag; Michael Antoniou; Steve Gilbert; Leonardo Trasande; Amy Itescu; Joseph Allen
Subject: Re: The Shredding of Exponent

Excellent. Thanks for sending

> On Dec 13, 2016, at 9:58 AM, Pete Myers <jpmyers@ehsic.org> wrote:
>
> worth reading:
>
> Dec 13 >From asbestos to pesticides to pork. Big companies in legal scrapes turn to science-for-hire giant Exponent. **FairWarning**.

From: Patricia Hunt <pathunt@vetmed.wsu.edu>
To: Pete Myers <jpmyers@ehsic.org>
Sent: 12/15/2014 10:24:39 AM
Subject: Re: Theo

Attach: [EMB4_theo-final.jpg]
Hi Pete-

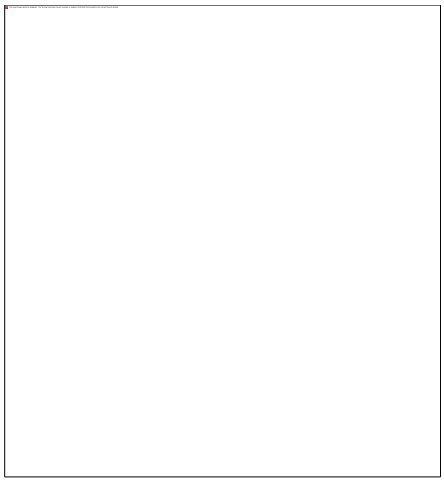
Thanks for sending this. Losing Theo is a major loss for all of us, but I know it will be particularly hard on you. She was a rare and unusual woman but I think her spirit touched everyone she met. Thus, I hope you draw comfort from knowing that a little bit of Theo carries on in each of us. May we all strive to put less of our ego and more of our love into everything we do!

With warm hugs,

Pat

From: Pete Myers <jpmyers@ehsic.org>
Date: Monday, December 15, 2014 9:54 AM
To: "Karp, Harvey" <montee@earthlink.net>, "Prins, Gail" <gprins@uic.edu>, "Lanphear, Bruce" <blanphear@sfu.ca>, "Cranmer, Joan" <cranmerJoanM@uams.edu>, "Cory-Slechta, Deborah" <deborah_cory-slechta@urmc.rochester.edu>, Peter Orris <porris@uic.edu>, Fred Vom Saal <vomsaalF@missouri.edu>, Terry Collins <tc1u@andrew.cmu.edu>, Howard Snyder <snyderh@email.chop.edu>, Peter DeFur <pldefur@igc.org>, "Ho, Shuk-mei" <shuk-mei.ho@uc.edu>, "R. Thomas Zoeller" <tzoeller@bio.umass.edu>, "Prof. Louis J. Guillette" <lou.guillette@gmail.com>, Ted Schettler <tschettler@igc.org>, "Ozonoff, David" <dozonoff@bu.edu>, "Hayes, Tyrone" <tyrone@berkeley.edu>, "Woodruff, Tracey" <WoodruffT@obgyn.ucsf.edu>, "Dr. Steve Heilig" <heilig@sfms.org>, "Stahlhut, Richard" <richard_stahlhut@urmc.rochester.edu>, Sheldon Krimsky <sheldon.krimsky@tufts.edu>, Philip Landrigan <phil.landrigan@mssm.edu>, "Hunt, Patricia Ann" <pathunt@wsu.edu>, Shanna Swan <shanna.swan@mssm.edu>, "Hauser, Russ" <rhauser@hohp.harvard.edu>, Bruce Blumberg <blumberg@uci.edu>, Amy Kostant <amy@sciencecom.org>, Bernard Weiss <Bernard_Weiss@urmc.rochester.edu>, Kalee Kreider <kaleekreider@gmail.com>, Laura Vandenberg <lvandenberg@schoolph.umass.edu>, Emily Copeland <emily@sciencecom.org>, Carl-Gustaf Bornehag <caguborn@kau.se>, Michael Antoniou <michael.antoniou@kcl.ac.uk>, Steve Gilbert <sgilbert@innd.org>, Leonardo Trasande <leonardo.trasande@nyu.edu>, Amy Itescu <itescua@UCMAIL.UC.EDU>
Subject: Theo

Theo Colborn, 19272014



2014 Julie Dermansky for Earthworks

If you ever had the chance to meet her, even once, you knew Theo Colborn. She didnt have a single hidden agenda. Her commitment to uncovering the truth was out there for the world to see.

For nearly 30 years she dedicated herself to revealing the dangers of endocrine disrupting chemicals to wildlife and humans. More recently she alerted us all to the threats posed by chemicals associated with oil and gas development. She wove the two together beautifully in her statement **The Fossil Fuel Connection**, which she worked on until the day she died.

Theos visionary leadership and passion shone most brilliantly when she made direct connections between new ideas, scientists whose work confirmed them, impacted individuals, and people in positions to change what needed changing. She will be remembered for many generations to come, generations that she worked tirelessly to protect.

Theo often feared that we had already passed the tipping point that our intelligence and compassion had been so compromised by endocrine disruptors that we could no longer think our way out of the crises we had created.

As the living embodiment of her legacy, we at TEDX say, No. It is not too late. There are people out there who get it and who care a lot of people and we wont let you down Theo.

From the Staff and Board of Directors of TEDX

Theos family has requested that in lieu of flowers, donations be **sent to TEDX**.

Share your Theo Colborn story

Join our mailing list

Read a brief biography by Elizabeth Grossman

Read Theos CV

Keep fighting for the health and well being of all living things.

From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
To: Pete Myers <jpmyers@ehsic.org>
Sent: 1/2/2017 1:07:48 PM
Subject: Re: Time for a call?

Yes, we are back home and will be in at work tomorrow.

On 1/2/17, 10:47 AM, "Pete Myers" <jpmyers@ehsic.org> wrote:

>Are you back at WSU?

From: <JPMyers@ehsic.org>
To: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Sent: 7/29/2015 12:10:50 PM
Subject: Re: Today's call at noon ET

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awesome!

Pete Myers, from a mobile phone

On Jul 29, 2015, at 3:00 PM, Hunt, Pat <pathunt@vetmed.wsu.edu> wrote:

> Hi Pete-
>
> I am really sorry that I could not make the call this week. I am interested in the direction you propose because I am getting tired of the constant battles with industry. The time has come for a much larger picture focus and a mission to educate! We are working on this on our campus as a way of moving reproductive sciences back into the spotlight by focusing not just on reproduction but on the fact that most human diseases have their genesis in the womb. Count me in!
>
> Pat

From: Pete Myers <jpmyers@ehsic.org>
Date: Wednesday, July 29, 2015 at 8:38 AM
To: Harvey Karp <dr.karp@thehappiestbaby.com>, "Prins, Gail" <gprins@uic.edu>, "Lanphear, Bruce" <blanphear@sfu.ca>, "Cranmer, Joan" <cranmerJoanM@uams.edu>, "Cory-Slechta, Deborah" <deborah_cory-slechta@urmc.rochester.edu>, Peter Orris <porris@uic.edu>, Fred Vom Saal <vomsaalF@missouri.edu>, Terry Collins <tc1u@andrew.cmu.edu>, Howard Snyder <snyderh@email.chop.edu>, Peter DeFur <pldefur@igc.org>, "Ho, Shuk-mei" <shuk-mei.ho@uc.edu>, "R. Thomas Zoeller" <tzoeller@bio.umass.edu>, "Prof. Louis J. Guillette" <lou.guillette@gmail.com>, "Ozonoff, David" <dozonoff@bu.edu>, "Hayes, Tyrone" <tyrone@berkeley.edu>, "Woodruff, Tracey" <WoodruffT@obgyn.ucsf.edu>, "Dr. Steve Heilig" <heilig@sfms.org>, "Stahlhut, Richard" <richard_stahlhut@urmc.rochester.edu>, Sheldon Krimsky <sheldon.krimsky@tufts.edu>, Philip Landrigan <phil.landrigan@mssm.edu>, "Hunt, Patricia Ann" <pathunt@wsu.edu>, Shanna Swan <shanna.swan@mssm.edu>, Russ Hauser <rhauser@hohp.harvard.edu>, Bruce Blumberg <blumberg@uci.edu>, Amy Kostant <amy@sciencecom.org>, Bernard Weiss <Bernard_Weiss@urmc.rochester.edu>, Kalee Kreider <kaleekreider@gmail.com>, Laura Vandenberg <lvandenberg@schoolph.umass.edu>, Emily Copeland <emily@sciencecom.org>, Carl-Gustaf Bornehag <caguborn@kau.se>, Michael Antoniou <michael.antoniou@kcl.ac.uk>, Steve Gilbert <sgilbert@innd.org>, Leonardo Trasande <leonardo.trasande@nyu.edu>, Amy Itescu <itescua@UCMAIL.UC.EDU>
Cc: Emily Copeland <emily@sciencecom.org>
Subject: Today's call at noon ET

Planetary Boundaries

Id like to briefly solicit reactions today to a new thrust we are exploring at Environmental Health Sciences/Environmental Health News.

Ive spoken about this with several of you already, so please excuse the repetition.

Beginning in 2009 a group of scientists at the Stockholm Resilience Institute (SRI) began publishing about a framework that organizes looming planet scale environmental challenges around a set of planetary boundaries. Ive attached the original piece from Nature as well as a 2015 publication from Science. In addition, I have included two papers that build off of these, one specifically focused on toxicity and one on planetary health.

SRI proposes that a set of forces they call planetary boundaries exist that human activities have been pushing toward limits (boundaries) beyond which their impacts call into question the ability of civilization to thrive.

They are diagramed in the graph here, taken from their February 2015 paper in Science, attached.

Note that there are three boundaries with question marks. One of them, novel entities, includes the impacts of toxic chemicals [novel entities is a term apparently forced upon them by the editors of Science who did not want chemicals or nano mentioned explicitly (I am told this by an independent observer party to confidential information)].

SRI acknowledges they dont have the expertise to handle toxic chemicals. One group of experts, including īe Bergman and Martin Scheringer, published a brief commentary, attached, that reiterates the importance of this boundary but really doesnt make much progress beyond that.

I want to explore with those of you who are interested how we might advance this issue: How at either regional or global levels could toxification play a boundary role?

This issue is of importance for several reasons. First, it is simply an important question to ask.

Second, by developing information about toxification as a planetary boundary within the SRI framework (which we think needs some additional refinement) we legitimate the importance of toxification relative to the other issues. Currently, climate is grabbing the worlds attention. It should, but other things are going on that are vitally important, including toxification, and they all may interact (heres a link to a great recent essay by Margaret Atwood titled Its not climate change its everything change. http://bit.ly/1SbOkYE)

Further, and from an institutional perspective, I think that by placing toxification within the SRI framework, we gain access to funding institutions who wont fund work solely on that but would on the overall framework. See the reprint, sent separately, from Lancet funded by the Rockefeller Foundation on planetary health, which takes the SRI framework and modifies it (they do more than SRI did on toxification but fall far short of where it might go.

How to proceed?

I propose to convene a group of people who would be willing to brainstorm on this, first by phone then in person in a workshop.

For example, would region-wide lead poisoning via its effect on impulse control lead to areas of civil dysfunction that could render civil society unmanageable?

Or would the contribution of metabolic disruptors elevate levels of obesity and type 2 diabetes to such rates that national health care systems are bankrupted, with widespread consequences for all health?

Or could fertility rates caused by EDCs fall so low that the age structure of a nations population became so shifted toward aging geezers that social security systems go bankrupt.

Could widespread toxification so disrupt pollination that food security was undermined (affecting functional biodiversity)?

Whether these or correct or not is not the point. I want to bring together a group of really smart, creative people who will be willing to spend some serious time generating ideas, evaluating them and then writing it up.

Here at EHS/EHN we already have been playing with a new way to organize EnvironmentalHealthNews.org. It is based upon a modified version of the SRI framework, simplified and coalesced into bites that arent too technical for our audience.

You can see a draft at ehn.ehstest.org (account 41 password 41). Please dont share that. Key to this are the section fronts just beneath the masthead that read from left to right Front page, Toxification, Children etc. Click on them.

We are still working to make this better. This is a baby step that we were able to implement without developing very much new web engineering. There will be several iterations to follow.

I close by guaranteeing that The Donald and The Donald Cat do not figure in our plans.

<2015-0203 Planetary boundaries.png>

<2015-0728 The Donald Cat.png>

From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
To: Pete Myers <JPMyers@ehsic.org>
Sent: 7/29/2015 12:00:20 PM
Subject: Re: Today's call at noon ET

Attach: [EMB4_2015-0203 Planetary boundaries.png] [EMB4_2015-0728 The Donald Cat.png]
Hi Pete-

I am really sorry that I could not make the call this week. I am interested in the direction you propose because I am getting tired of the constant battles with industry. The time has come for a much larger picture focus and a mission to educate! We are working on this on our campus as a way of moving reproductive sciences back into the spotlight by focusing not just on reproduction but on the fact that most human diseases have their genesis in the womb. Count me in!

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Here at EHS/EHN we already have been playing with a new way to organize EnvironmentalHealthNews.org. It is based upon a modified version of the SRI framework, simplified and coalesced into bites that arent too technical for our audience.

You can see a draft at ehn.ehstest.org (account 41 password 41). Please dont share that. Key to this are the section fronts just beneath the masthead that read from left to right Front page, Toxification, Children etc. Click on them.

We are still working to make this better. This is a baby step that we were able to implement without developing very much new web engineering. There will be several iterations to follow.

I close by guaranteeing that The Donald and The Donald Cat do not figure in our plans.

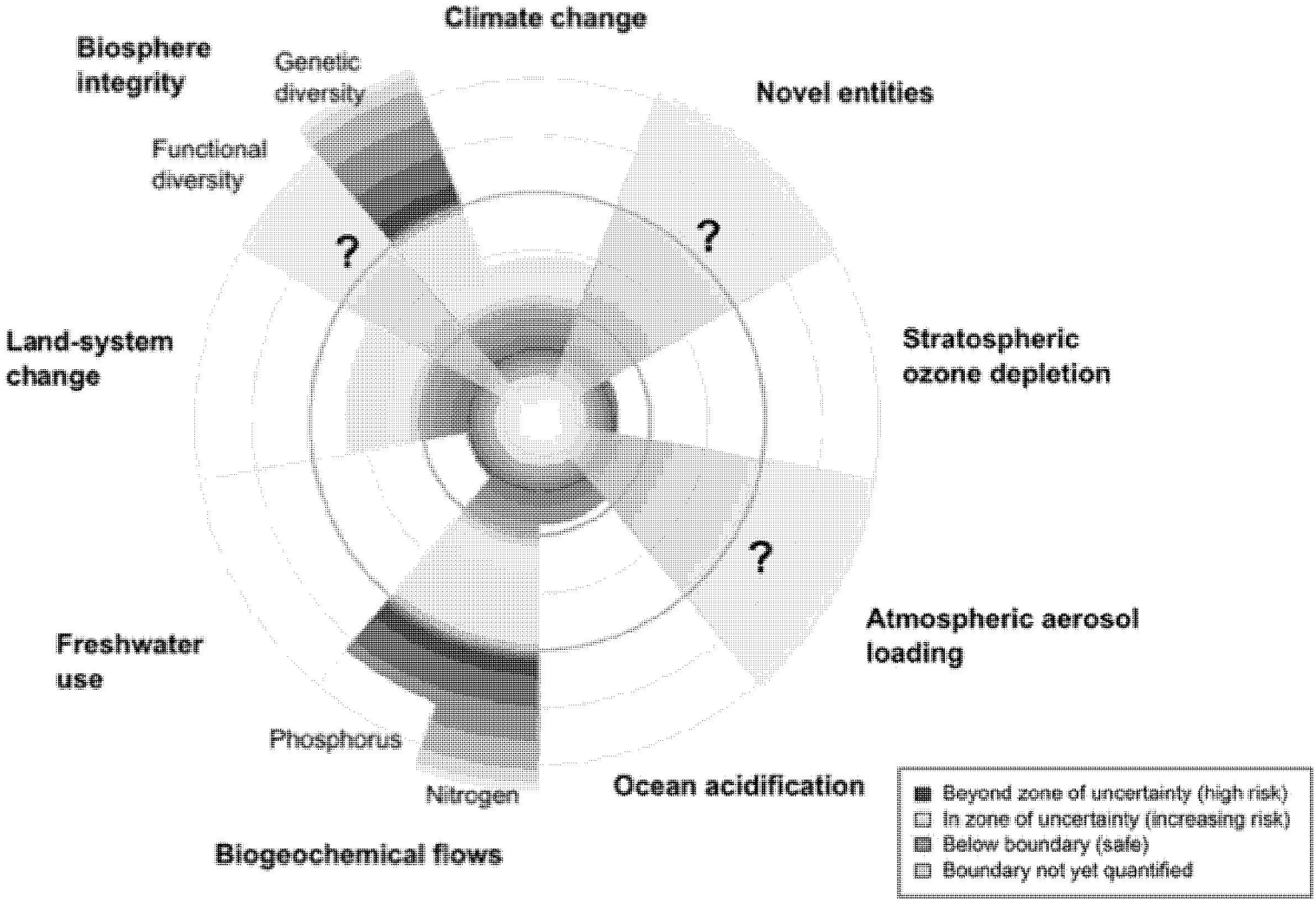

Fig. 3. The current status of the control variables for seven of the nine planetary boundaries. The green zone is the safe operating space (below the boundary), yellow represents the zone of uncertainty (increasing risk), and red is the high-risk zone. The planetary boundary itself lies at the inner heavy circle. The control variables have been normalized for the zone of uncertainty (between the two heavy circles); the center of the figure therefore does not represent values of 0 for the control variables. The control variable shown for climate change is atmospheric CO_2 concentration. Processes for which global-level boundaries cannot yet be quantified are represented by gray wedges; these are atmospheric aerosol loading, novel entities, and the functional role of biosphere integrity. Modified from (*1*).

From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
To: Amy Kostant <amy@sciencecom.org>
Sent: 6/22/2017 7:04:16 PM
Subject: Re: tomorrow

That would be perfect - as long as I get to buy you lunch! I can see it on my map and getting there will be easy. I'll be there shortly after 1:30, but don't rush - I will be catching up on The NYTimes.

Sent from my iPad

On Jun 22, 2017, at 9:35 PM, Amy Kostant <amy@sciencecom.org> wrote:

> Hi Pat,
> Ill be in Rockville until about 1:00, but I can meet you in Tysons easily by 1:30 -2:00. How about Caf頃eluxe for a late lunch?
> If you prefer to come into DuPont because you want to get out of Tysons, thats fine too. For me, its equal.
>
> ---
>
> **From:** Hunt, Pat [mailto:pathunt@vetmed.wsu.edu]
> **Sent:** Thursday, June 22, 2017 9:32 PM
> **To:** Amy Kostant
> **Subject:** tomorrow
>
> Hi Amy-
>
> They say we should be finished by 12:30 or so tomorrow. It sounds like it will be rainy and humid let me know what works best for you. I am not adverse to hopping the metro and meeting you.
>
> Pat

From: Amy Kostant <amy@sciencecom.org>
To: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Sent: 6/22/2017 7:39:28 PM
Subject: RE: tomorrow

Im so glad that works. But we split it.
Im looking forward to seeing you!

From: Hunt, Pat [mailto:pathunt@vetmed.wsu.edu]
Sent: Thursday, June 22, 2017 10:04 PM
To: Amy Kostant
Subject: Re: tomorrow

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From: Amy Kostant <amy@sciencecom.org>
To: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Sent: 6/22/2017 6:35:01 PM
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From: Patricia Hunt <pathunt@vetmed.wsu.edu>
To: Amy Kostant <amy@sciencecom.org>
Sent: 7/25/2014 12:54:32 PM
Subject: Re: tough questions call with you and Terry

Hi Amy-

Sorry, things have been really crazy here this week. Yes, next Tuesday at 2pm eastern time is fine. I'm glad that she did well. If she talks to me, I will try to get her to use my stuff to talk beyond the paper results.

Pat

From: Amy Kostant <amy@sciencecom.org>
Date: Thursday, July 24, 2014 1:21 PM
To: Patricia Hunt <pathunt@vetmed.wsu.edu>
Cc: Emily Copeland <emily@sciencecom.org>
Subject: tough questions call with you and Terry

Hi Pat
I just did media trailing with Terry. She asked great questions and I think shell do fine. I suggested she talk with you about how much she can say with regard to the second paper.
Could you join a call next Tuesday at 2 pm eastern to talk through tough questions with Terry, Emily and me? If not, do you have anytime avaialbe that afternoon?
Cheers -
Amy

****
Amy Kostant
Science Communication Network
Office: 301-654-6665
Cell: 202-255-6665
amy@sciencecom.org

From: Patricia Hunt <pathunt@vetmed.wsu.edu>
To: Pete Myers <jpmyers@ehsic.org>
Sent: 2/26/2014 10:40:22 AM
Subject: Re: Tracey's on board

Attach: [EMB4_2014-0218 Greater Flamingo march.jpg]
What an awesome photo! How did you get them to organize by color?

From: Pete Myers <jpmyers@ehsic.org>
Date: Wednesday, February 26, 2014 10:27 AM
To: Patricia Hunt <pathunt@vetmed.wsu.edu>
Subject: Re: Tracey's on board

Thank you!

Just got back from Dubai. Discovered they have living lawn ornaments

On Feb 26, 2014, at 1:24 PM, Hunt, Pat <pathunt@vetmed.wsu.edu> wrote:

Hi Pete-

Tracey is willing. I wrote Fred and asked him to send me talking points. If the others do the same, I would like to write a rough draft tomorrow. I figure it will be easier to organize a call around a rough draft.

Pat

From: <JPMyers@ehsic.org>
To: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Sent: 12/7/2015 11:43:08 AM
Subject: Re: UPDATE!

---

so we are taking your counsel. Davenport it is! We'll see you tomorrow morning.

Pete Myers, from a mobile phone

On Dec 7, 2015, at 11:21 AM, Hunt, Pat <pathunt@vetmed.wsu.edu> wrote:

> It might be possible to catch an earlier flight as there are nearly hourly flights between Seattle and Spokane. If you end up arriving late, you could stay at the Davenport. It is a lovely old hotel with great food. It may be pricier than youd like, but it is a treat.
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>
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> **From:** Pete Myers <jpmyers@ehsic.org>
> **Date:** Monday, December 7, 2015 at 11:04 AM
> **To:** Patricia Hunt <pathunt@vetmed.wsu.edu>
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> Date: 12/07/2015 01:56PM
> Subject: Re: UPDATE!
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Date: Monday, December 7, 2015 at 10:11 AM
To: patricia hunt <pathunt@vetmed.wsu.edu >
Subject: UPDATE!

update: we land at Spokane at 5:58! Is dinner still possible? We'll have a rental.
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To: "JPMyers@ehsic.org" < JPMyers@ehsic.org>
From: "Hunt, Pat" < pathunt@vetmed.wsu.edu>
Date: 12/07/2015 11:57AM
Subject: Re: Hi Pat

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Sent from my iPhone

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From: "Hunt, Pat" < pathunt@vetmed.wsu.edu >
Date: 11/30/2015 01:39PM
Subject: Re: Hi Pat

Hi Pete-

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Yes, the 7th/8th still works. It is a long drive from Portland but there
are some very interesting parts. I doubt that you will get here too early
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be easier and more relaxing for the two of you to have dinner at my house.
Neither Pullman or Moscow is known for fine dining and I do enjoy cooking
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>Tell me if driving is crazy. I haven□□een up the Columbia River gorge
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From: <JPMyers@ehsic.org>
To: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Sent: 12/7/2015 11:04:53 AM
Subject: Re: UPDATE!

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To: "JPMyers@ehsic.org" <JPMyers@ehsic.org>
Sent: 12/7/2015 11:50:49 AM
Subject: Re: UPDATE!

Most excellent plan! Tomorrow you will likely have both a large breakfast and a big lunch, but we can make lunch late. The drive from my house to the airport is an easy 15 min and the airport is small, so being there by 3 would give you plenty of time.

I am glad you decided not to rush. It is a long drive and doing it after dark and in the rain is a real pain.

Enjoy your night!

From: Pete Myers <jpmyers@ehsic.org>
Date: Monday, December 7, 2015 at 11:43 AM
To: patricia hunt <pathunt@vetmed.wsu.edu>
Subject: Re: UPDATE!

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>in decades. And I haven□□een in eastern Washington since 1972.

From: Patricia Hunt <pathunt@vetmed.wsu.edu>
To: "JPMyers@ehsic.org" <JPMyers@ehsic.org>
Sent: 12/7/2015 11:21:44 AM
Subject: Re: UPDATE!

It might be possible to catch an earlier flight as there are nearly hourly flights between Seattle and Spokane. If you end up arriving late, you could stay at the Davenport. It is a lovely old hotel with great food. It may be pricier than youd like, but it is a treat.

Lunch would be fun either at my house (if we miss dinner tonight) or in town. Id like you to meet Margrit von Braun. She is the daughter of Wernher and a scientist herself (she is an environmental engineer). She is also a member of Collegium Ramazinni and the most active retired professor I have ever met. I think she might fit into your plans. She is traveling back from Moses Lake tomorrow and isnt sure if she will make it in time for lunch. If she does, do you mind if I include her in our lunch plans?

From: Pete Myers <jpmyers@ehsic.org>
Date: Monday, December 7, 2015 at 11:04 AM
To: Patricia Hunt <pathunt@vetmed.wsu.edu>
Subject: Re: UPDATE!

We return via Pullman. So lunch would be possible. I must admit this has been unusually stressful juggling all series of delayed flights, missed connections and cancelled flights. Thanks for your understanding. I'll keep you up to date!

-----"Hunt, Pat" <pathunt@vetmed.wsu.edu> wrote: -----
To: "JPMyers@ehsic.org" <JPMyers@ehsic.org>
From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Date: 12/07/2015 01:56PM
Subject: Re: UPDATE!

I was just writing you and telling you not to worry about dinner and that I would understand if you chose to bail on the whole thing. The drive from Spokane is about an hour and a half. Id be happy to have a late dinner, but I dont want you to drive in the rain and after dark if you are tired (its not the safest highway and I hate driving it in the dark). Lets stay loose and see what happens. If you end up staying in Spokane, I will give you some good dinner ideas. If you make it down here fabulous. Remember, we can always do lunch tomorrow. Although that wont work if you are flying out of Spokane at 3:45!

From: Pete Myers < jpmyers@ehsic.org>
Date: Monday, December 7, 2015 at 10:11 AM
To: patricia hunt <pathunt@vetmed.wsu.edu >
Subject: UPDATE!

update: we land at Spokane at 5:58! Is dinner still possible? We'll have a rental.
-----"Hunt, Pat" < pathunt@vetmed.wsu.edu> wrote: -----

To: "JPMyers@ehsic.org" < JPMyers@ehsic.org>
From: "Hunt, Pat" < pathunt@vetmed.wsu.edu>
Date: 12/07/2015 11:57AM
Subject: Re: Hi Pat

No, not if the plane can land - flying in and out of Pullman in the winter is always tricky. Shall we pick you up at the airport?

Sent from my iPhone

On Dec 7, 2015, at 6:53 AM, " JPMyers@ehsic.org" <JPMyers@ehsic.org > wrote:

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> Date: 12/07/2015 09:23AM
> Subject: Re: Hi Pat
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> I am glad to hear that you are not driving, the weather is pretty miserable (although we certainly cant complain about rain!). Travel safely and keep me posted.
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> Pat
>
> ---
>
> **From:** Pete Myers < jpmyers@ehsic.org>
> **Date:** Sunday, December 6, 2015 at 9:36 PM
> **To:** patricia hunt <pathunt@vetmed.wsu.edu >
> **Subject:** Re: Hi Pat
>
> We had some glitches in our travel schedule today so didn't reach Portland yet. We'll be there by tomorrow evening for sure. Probably not driving. As soon as I know what's happening I'll let you know. We will get rooms in Moscow for tomorrow night. And unless something unexpected happens we should be there for dinner.
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> To: Pete Myers <jpmyers@ehsic.org >
> From: "Hunt, Pat" < pathunt@vetmed.wsu.edu >
> Date: 11/30/2015 01:39PM
> Subject: Re: Hi Pat

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I□□orry I missed your call, we went to Portland for the holiday.

Yes, the 7th/8th still works. It is a long drive from Portland but there
are some very interesting parts. I doubt that you will get here too early
in the afternoon, but we should definitely plan to have dinner. It might
be easier and more relaxing for the two of you to have dinner at my house.
Neither Pullman or Moscow is known for fine dining and I do enjoy cooking
- although I would need to know about dietary restrictions for both of
you. Alternatively, you may wish to extend your trip by taking advantage
of good restaurants and fine wine along the way (e.g., Walla Walla), and
that is fine too.

Wow, do I understand the comment about rooms that are not heavily scented.
I always request a room for someone with chemical sensitivities but that
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To: "JPMyers@ehsic.org" <JPMyers@ehsic.org>
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From: Amy Kostant <amy@sciencecom.org>
To: Bruce Blumberg <blumberg@uci.edu>, jerry heindel <jerryheindel@gmail.com>, "R. Thomas Zoeller" <tzoeller@bio.umass.edu>, Pete Myers <jpmyers@ehsic.org>, Fred Vomsaal <VomsaalF@missouri.edu>, "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Sent: 8/23/2017 8:32:58 AM
Subject: RE: updated program from jerry

Hi all,
A few thoughts based on the program agenda:

- Weve purposely invited people who arent researchers, so should the focus question (first break out) reflect more of the expertise of the attendees? I want to be sure everyone participates from the beginning.
- Can someone provide details on what the Plenary: Whats in a name? will address?
- I think we discussed inviting some younger folks. Chris Kassotis? And perhaps another NGO Nsedu Obot Witherspoon, MPH, who runs CEHN?

Amy

From: Bruce Blumberg [mailto:blumberg@uci.edu]
Sent: Tuesday, August 15, 2017 1:40 PM
To: jerry heindel; R. Thomas Zoeller
Cc: Pete Myers; Fred Vomsaal; Amy Kostant; Hunt, Pat
Subject: Re: updated program from jerry

2 or 3 breakouts, whichever works for the agenda. If 2, I'd rather not have on the same day because having the sessions and reporting on them properly takes adequate time and energy.

On 8/15/2017 9:55 AM, jerry heindel wrote:

> Of course there will be funds for the rooms so we can have the best meeting we want. If you want 4 breakouts some on each daywe can do that. If we dont need 4 breakouts then we can have 2 on same day and save money. Saving money is not the goalbut no need to waste money.
>
> I just need to know if we want breakouts on both days or not.
>
> Sent from Mail for Windows 10
>
> ---
>
> **From:** Bruce Blumberg
> **Sent:** Tuesday, August 15, 2017 12:40 PM
> **To:** jerry heindel; R. Thomas Zoeller
> **Cc:** Pete Myers; Fred Vomsaal; Amy Kostant; Hunt, Pat
> **Subject:** Re: updated program from jerry
>
> Must our agenda be driven by how many rooms are needed on which days? There must be a few hundred dollars sitting somewhere that can pay for meeting rooms in a hotel if we need them to accomplish our goals in the best way possible....

On 8/15/2017 9:02 AM, jerry heindel wrote:

> Thanks tom, are you ok with 2 breakouts on first day and then roundtables on second day? If so I just need breakout rooms for first day. jerry
>
> Sent from Mail for Windows 10
>
> ---
>
> **From:** R. Thomas Zoeller
> **Sent:** Tuesday, August 15, 2017 9:51 AM
> **To:** jerry heindel
> **Cc:** Bruce Blumberg; Pete Myers; Fred Vomsaal; Amy Kostant; Hunt, Pat
> **Subject:** Re: updated program from jerry
>
> Heres a couple of comments

From: Amy Kostant <amy@sciencecom.org>
To: jerry heindel <jerryheindel@gmail.com>, 'Pete Myers' <jpmyers@ehsic.org>, "R. Thomas Zoeller" <tzoeller@Bio.umass.edu>, "Prof. Fred vom Saal" <VomsaalF@missouri.edu>, Bruce Blumberg <blumberg@uci.edu>, "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Sent: 7/23/2017 7:04:18 AM
Subject: RE: updated version of global EDC workshop program

Attach: [Endocrine Disruption Strategies Workshop v2 ak.docx]
Hi All,
Apologies I didnt attach this in my earlier email.
Amy

From: Amy Kostant
Sent: Friday, July 21, 2017 4:17 PM
To: 'jerry heindel'; 'Pete Myers'; R. Thomas Zoeller; Prof. Fred vom Saal; Bruce Blumberg; Hunt, Pat
Subject: RE: updated version of global EDC workshop program

Hi All,
Ive added a few comments.
Amy

From: jerry heindel [mailto:jerryheindel@gmail.com]
Sent: Wednesday, July 19, 2017 5:10 PM
To: 'Pete Myers'; R. Thomas Zoeller; Prof. Fred vom Saal; Amy Kostant; Bruce Blumberg; Hunt, Pat
Cc: jerry heindel
Subject: updated version of global EDC workshop program

Thanks everyone for your helpful commentsand your quick reply. I realize you all have day jobs

I tried to improve the program but have some questions that I put in the program. I think it is moving in the right direction. Need to focus on the most important key issues and leave sufficient time for lots of discussion and socializing.

If I get comments in time I can develop another draft before our next meeting time is getting short. If any of you want to call and chat about the program individually or in groups I can do that too. I have lots of time

We will have our next call on Friday July 28th at 1 pm. 605 475 2875 code [redacted]

Sent from Mail for Windows 10

Endocrine Disruption Strategies Workshop

December 4-5, 2017

Hotel in Raleigh NC (TBD)

Introduction/Goa :

The EDC topic is particularly challenging because it encompasses sub-topics from molecular physiology and endocrinology to genetics and personalized medicine, economics and sociology. Studies of EDCs have and will continue to provide key data that can improve health but the impact on health will only be as successful as we are at communicating the findings and implications to clinicians, general public as well as chemists, lawyers, regulators and policy makers. Many people and working groups have become interested in the effects of EDCs including but not limited to the important work of SCN and EHN, CHE, TEDX, EDCfree and others in the NGO community, the Endocrine Society and many more around the globe. Nonetheless the study of EDCs is not a discipline per se but a topic that crosses many disciplines. Indeed, the study of EDCs has provided biological insight in a number of different fields. We propose that the study of EDCs could be strengthened by the development of a yearly forum that increases communication and collaboration across disciplines to both inform the science and expand its impact on human and ecological health.

The goal of this workshop is thus to set up a yearly forum (brainstorming/planning session workshop) to help identify strategic needs and opportunities to advance the field and encourage~~s~~ strategic collaborations. This forum will provide a platform to define the critical issues facing the field, and develop plans to improve knowledge sharing, coordination and collaboration that will reduce the impact of EDC exposures on human health and improve the impact of EDC science on the regulation of EDCs.

The initial meeting will focus on key areas important to the EDC field including, efforts to bolster consumer interest in safer products, collaboration with chemists on chemical design, research needs and opportunities, improved coordination/collaboration among NGOs and regulatory and policy needs and coordination with lawyers around lawsuit opportunities. There are specific needs in the EU centered around EDC regulations/guidelines and in the US centered around the Administration's focus on undermining environmental policies and the emergence of science deniers. The field also needs to build/strengthen ties between medical professional groups whose health goals are impaired by EDCs. The ~~general goal~~general goal is to "see" where the field is, understand how it got there and ~~how~~determine how to move forward to improve our ability to reduce disease from environmental exposures. It will also focus on preparing for the next Forum which we propose will take place after the EDC Gordon Conference in Switzerland.

Planning Committee: Jerry Heindel, Pete Myers, Amy Kostant, Fred vom Saal, Bruce Blumberg, Tom Zoeller, Pat Hunt, Joe DiGangi

Commented [AK1]: Who is the audience for this? I'm wondering if they'll know all the acronyms.

Commented [jh2]: This is nice to say but I don't see how we can cover all these areas in the meeting. Perhaps an overview talk presenting all the challenges and opportunities to set the stage…then focus on some of most important ones in the meeting…what would those be…so there is sufficient time for discussion to come to some conclusions and ideas for a path forward.

Commented [AK3]: Would it work to think in terms of how the outcomes will be used – by public health agencies; lawmakers; consumer campaigns; to combat science deniers; …

Program Overview:

Sunday evening, reception at Jerry Heindel's House

Monday, December 4th

8:30 Welcome, introduction and overview of the meeting

9:00 **Plenary Talk:** What is in a name: Signal toxicity, metabolism disruptors, obesogens or EDCs

9:30-11:00 **Breakout groups**

1. Significant impacts of EDCs on human health: major accomplishments/ how were they accomplished/lessons learned that will help future activities
2. EDC research needs to improve impact of research/ new approaches/technologies
3. What are the big health challenges in endocrinology that have not yet been examined through and EDC lens and how might that happen?

11:00 Report of breakout groups

Lunch

1:30 Plenary: Global EDC policy issues : EU vs USA

2:30 Breakout Sessions:

1. Challenges/opportunities related to risk assessment, guideline studies and AOPs for EDCs
2. How do we develop a unified multinational response to attacks on scientific integrity from various sources?
3. How to build/expand ties between medical professional groups whose health goals are impaired by EDCs.
4. How to change science to aid in science litigation

4:00 Breakout reports/Discussion

5:15 End for day…

Dinner at Hotel or restaurant at Crabtree Mall, (0.5 miles)

Tuesday December 5th

8:30 **Plenary**: Overview of goals and plans of Advocacy Groups/ NGOs and Societies

TEDX, CHE, SCN, EHN, EWG, PHRE, EDCFree, Endocrine Society (what are the main key ones to focus on here?) 15 min each

10:15 Break

10:30 **Breakouts**: How to improve the knowledge and acceptance of EDC data/principles/effects on human and wildlife health

Commented [jh4]: I like Pat's thought on …we need to invent some type of structure for moving far beyond the science. I don't understand that? Tell me more and put that someplace on the program.

Commented [AK5]: Not sure exactly what the question would be – what it is we want to know, but given the people invited we might explore the role of: NGOs, health affected communities, and science communicators in moving the field forward. (How they'll use the science – what they need to move the science into public action.)

Commented [jh6]: Pete had a comment on setting up groups to brainstorm on a topic of their choosing… I need to know more about this idea. The breakouts are meant to contain people from varied backgrounds brainstorming on a specific topic.

1. How can NGOs and advocacy groups better communicate and coordinate their messages?
2. How do we expand into new avenues of communication, social media, infographics, etc.
3. How to Communicate with and gain acceptance of scientific data by community, clinicians, science deniers? How do we change peoples' thinking?

Commented [AK7]: This may not be the right task for this meeting. Perhaps instead we should ask the NGOs what they need from scientists in order to make their campaigns more effective.

12:00 Lunch

1:15 Reports from Breakouts

3:00 Break

3:20 **Plenary Session**: Moving Forward…General Discussion of next EDC Forum

- Can we develop working groups/focused workshops? Or listservs? What would be their purpose/goals?
- Plan for the next year's meeting… Switzerland after Gordon Conference…need planning committee
- Discussion of meeting output

(POSSIBLE)Attendees (by invitation only) Everyone pays all own expenses

Commented [AK8]: Do we want someone from a journal – EHP?

Jerry Heindel
Bruce Blumberg
Fred vom Saal
Tom Zoeller
Pat Hunt
Jodi Flaws
Russ Hauser

Formatted: Spanish (Mexico)

Joe DiGangi
Leo Trasande
Shanna Swan
Gail Prins
Ana Soto
Robert Sargis
Matt Cave
Philippe Grandjean
Linda Birnbaum
Frank von Hippel
Heather Patisaul
Andrea Gore
David Crews
Linda Giudice
Laura Vandenberg
Susan Jobling

Juliette Legler
Andreas Kortenkamp
Ake Bergman
Angel Nadal
Tracy Woodruff
Shirlee Tan
Joe Laasko
Michael Lerner
Ken Cook
Terry Collins
Carol Kwiatkowski
Karen Wang
Jeff Wise
Shorey Myers
Barbara Demeniex
Mike Schade
John Pierre Bourguignon
Niels Skakkebaek
Arlene Blum
Ninja Reineke
Gwynne Lyons
Jane Muncke
Jun Kanno
Loretta Doan

Expertise in Regulatory and Policy: Pete Myers, Tracy Woodruff, Shirlee Tan, Heather Patisaul, Tom Zoeller, Joe Laasko, Joe DiGangi, Michael Lerner, Ken Cook, Linda Birnbaum

Expertise in Green Chemistry: Terry Collins

Expertise in Communications: Amy Kostant, Pete Myers, Carol Kwiatkowski, Tracy Woodruff, Karen Wang, Jeff Wise, Pat Hunt, Jane Muncke

NGO community/Scientific Societies: EHN, EWG, TEDX, CHE, Endocrine Society, SCN, PHRE, PEDS, Heal, Chemtrust, HEFN, community action groups, Mind the Store(safer chemicals: healthy families), Arlene Bloom, SEHN (Ted Schecter), EDF, Non toxic Irvine (others ?)

Expertise in Funding: Michael Lerner, Shorey Myers, Pete Myers, Linda Birnbaum, Jeff Wise

Representative from EDC Gordon Conference Planning (June 2-8 Les Diablerets Switzerland): Jodi Flaws

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Sent: 7/23/2017 12:31:05 PM
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---

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We will have our next call on Friday July 28th at 1 pm. 605 475 2875 code 41

Sent from Mail for Windows 10

<Endocrine Disruption Strategies Workshop v2 ak_bb.docx>

From: Pete Myers <jpmyers@ehsic.org>
To: Bruce Blumberg <blumberg@uci.edu>
CC: Amy Kostant <amy@sciencecom.org>, Jerry Heindel <jerryheindel@gmail.com>, "R. Thomas Zoeller" <tzoeller@Bio.umass.edu>, "Prof. Fred vom Saal" <VomsaalF@missouri.edu>, "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Sent: 7/23/2017 12:30:36 PM
Subject: Re: updated version of global EDC workshop program

Bruce thats great re Kano!

> On Jul 23, 2017, at 3:02 PM, Bruce Blumberg <blumberg@uci.edu> wrote:
>
> Happy Sunday everyone,
>
> I made a few additions and comments to Amy's version.
>
> Best,
>
> Bruce
>
> On 7/23/2017 7:04 AM, Amy Kostant wrote:
>
> > Hi All,
> > Apologies I didnt attach this in my earlier email.
> > Amy
> >
> > **From:** Amy Kostant
> > **Sent:** Friday, July 21, 2017 4:17 PM
> > **To:** 'jerry heindel'; 'Pete Myers'; R. Thomas Zoeller; Prof. Fred vom Saal; Bruce Blumberg; Hunt, Pat
> > **Subject:** RE: updated version of global EDC workshop program
> >
> > Hi All,
> > Ive added a few comments.
> > Amy
> >
> > **From:** jerry heindel [mailto:jerryheindel@gmail.com]
> > **Sent:** Wednesday, July 19, 2017 5:10 PM
> > **To:** 'Pete Myers'; R. Thomas Zoeller; Prof. Fred vom Saal; Amy Kostant; Bruce Blumberg; Hunt, Pat
> > **Cc:** jerry heindel
> > **Subject:** updated version of global EDC workshop program
> >
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From: Pete Myers <jpmyers@ehsic.org>
To: Jerry Heindel <jerryheindel@gmail.com>
CC: "R. Thomas Zoeller" <tzoeller@Bio.umass.edu>, "Prof. Fred vom Saal" <VomsaalF@missouri.edu>, Amy Kostant <amy@sciencecom.org>, Bruce Blumberg <blumberg@uci.edu>, "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Sent: 7/19/2017 2:24:51 PM
Subject: Re: updated version of global EDC workshop program

Attach: [2017-0719 JPM Endocrine Disruption Strategies Workshop v2.docx]
some quick thoughts

> On Jul 19, 2017, at 5:09 PM, jerry heindel <jerryheindel@gmail.com> wrote:
>
> Thanks everyone for your helpful commentsand your quick reply. I realize you all have day jobs
>
> I tried to improve the program but have some questions that I put in the program. I think it is moving in the right direction. Need to focus on the most important key issues and leave sufficient time for lots of discussion and socializing.
>
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>
> We will have our next call on Friday July 28th at 1 pm. 605 475 2875 code [redacted]
>
> Sent from Mail for Windows 10
>
> <Endocrine Disruption Strategies Workshop v2.docx>

Endocrine Disruption Strategies Workshop

December 4-5, 2017

Hotel in Raleigh NC (TBD)

Introduction/Goal:

The EDC topic is particularly challenging because it encompasses sub-topics from molecular physiology and endocrinology to genetics and personalized medicine, economics and sociology. Studies of EDCs have and will continue to provide key data that can improve health but the impact on health will only be as successful as we are at communicating the findings and implications to clinicians, general public as well as chemists, lawyers, regulators and policy makers. Many people and working groups have become interested in the effects of EDCs including but not limited to the important work of SCN and EHN, CHE, TEDX, EDCfree and others in the NGO community, the Endocrine Society and many more around the globe. Nonetheless the study of EDCs is not a discipline per se but a topic that crosses many disciplines. Indeed, the study of EDCs has provided biological insight in a number of different fields. We propose that the ~~study of~~ EDC field as a whole, including scientific understanding and its impact on reducing disease burden,s~~s~~ could be strengthened by the development of a yearly forum that increases communication and collaboration across disciplines ~~to both inform the science and expand its impact on human and ecological health.~~ .

The goal of this workshop is thus to set up a yearly forum (brainstorming/planning session workshop) to help identify strategic needs and opportunities to advance the field and encourages strategic collaborations. This forum will provide a platform to define the critical issues facing the field, and develop plans to improve knowledge sharing, coordination and collaboration that will reduce the impact of EDC exposures on human health and improve the impact of EDC science on the regulation of EDCs.

The initial meeting will focus on key areas important to the EDC field. The focus will be refined and sharpened over the coming months but might includ~~ing,e~~ efforts to bolster consumer interest in safer products, collaboration with chemists on chemical design, research needs and opportunities, improved coordination/collaboration among NGOs and regulatory and policy needs and coordination with lawyers around lawsuit opportunities. There are specific needs in the EU centered around EDC regulations/guidelines and in the US centered around the Administration's focus on undermining environmental policies and the emergence of science deniers. The field also needs to build/strengthen ties between medical professional groups whose health goals are impaired by EDCs. The general goal is to "see" where the field is, how it got there and how to move forward to improve our ability to reduce disease from environmental exposures. It will also focus on preparing for the next Forum which we propose will take place after the EDC Gordon Conference in Switzerland.

Commented [jh1]: This is nice to say but I don't see how we can cover all these areas in the meeting. Perhaps an overview talk presenting all the challenges and opportunities to set the stage…then focus on some of most important ones in the meeting…what would those be…so there is sufficient time for discussion to come to some conclusions and ideas for a path forward.

Planning Committee: Jerry Heindel, Pete Myers, Amy Kostant, Fred vom Saal, Bruce Blumberg, Tom Zoeller, Pat Hunt, Joe DiGangi

Program Overview:

Sunday evening, reception at Jerry Heindel's House

Monday, December 4th

8:30 Welcome, introduction and overview of the meeting

9:00 **Plenary Talk:** What is in a name: Signal toxicity, metabolism disruptors, obesogens or EDCs

Commented [jh2]: I like Pat's thought on …we need to invent some type of structure for moving far beyond the science. I don't understand that? Tell me more and put that someplace on the program.

9:30-11:00 **Breakout groups**

1. Significant impacts of EDCs on human health: major accomplishments/ how were they accomplished/lessons learned that will help future activities
2. EDC research needs to improve impact of research/ new approaches/technologies
3. What are the big health challenges in endocrinology that have not yet been examined through an~~d~~ EDC lens and how might that happen?

11:00 Report of breakout groups

Lunch

1:30 Plenary: Global EDC policy issues : EU vs USA

2:30 Breakout Sessions:

1. Challenges/opportunities related to risk assessment, guideline studies and AOPs for EDCs
2. How do we develop a unified multinational response to attacks on scientific integrity from various sources?
3. How to build/expand ties between medical professional groups whose health goals are impaired by EDCs.
4. ~~How to change science to aid in science litigation~~What opportunities are there to work with trial lawyers and how might EDC science be adjusted to maximize value here.

4:00 Breakout reports/Discussion

5:15 End for day…

Dinner at Hotel or restaurant at Crabtree Mall, (0.5 miles)

Tuesday December 5th

8:30 **Plenary**: Overview of goals and plans of Advocacy Groups/ NGOs and Societies

TEDX, CHE, SCN, EHN, EWG, PHRE, EDCFree, Endocrine Society (what are the main key ones to focus on here?) 15 min each

10:15 Break

10:30 **Breakouts**: How to improve the knowledge and acceptance of EDC data/principles/effects on human and wildlife health

1. How can NGOs and advocacy groups better communicate and coordinate their messages?
2. How do we expand into new avenues of communication, social media, infographics, etc.
3. How to Communicate with and gain acceptance of scientific data by community, clinicians, science deniers? How do we change peoples' thinking?

Commented [jh3]: Pete had a comment on setting up groups to brainstorm on a topic of their choosing… I need to know more about this idea. The breakouts are meant to contain people from varied backgrounds brainstorming on a specific topic.

Commented [JPM4]: It wasn't as broad as "of their choosing." It was to get each group to focus on a potential EDC campaign using science and communications and other tools of campaigns. Then leave time in the report-back session for evaluation of opportunities.

12:00 Lunch

1:15 Reports from Breakouts

3:00 Break

3:20 **Plenary Session**: Moving Forward…General Discussion of next EDC Forum

- Can we develop working groups/focused workshops? Or listservs? What would be their purpose/goals?
- Plan for the next year's meeting… Switzerland after Gordon Conference…need planning committee
- Discussion of meeting output

(POSSIBLE)Attendees (by invitation only) Everyone pays all own expenses
Jerry Heindel
Bruce Blumberg
Fred vom Saal
Tom Zoeller
Pat Hunt
Jodi Flaws
Russ Hauser
Joe DiGangi
Leo Trasande
Shanna Swan
Gail Prins
Ana Soto
Robert Sargis
Matt Cave
Philippe Grandjean
Linda Birnbaum
Frank von Hippel
Heather Patisaul
Andrea Gore

David Crews
Linda Giudice
Laura Vandenberg
Susan Jobling
Juliette Legler
Andreas Kortenkamp
Ake Bergman
Angel Nadal
Tracy Woodruff
Shirlee Tan
Joe Laasko
Michael Lerner
Ken Cook
Terry Collins
Carol Kwiatkowski
Karen Wang
Jeff Wise
Shorey Myers
Barbara Demeniex
Mike Schade
John Pierre Bourguignon
Niels Skakkebaek
Arlene Blum
Ninja Reineke
Gwynne Lyons
Jane Muncke
Jun Kanno
Loretta Doan
Leah Segedie (Mamavation)

Expertise in Regulatory and Policy: Pete Myers, Tracy Woodruff, Shirlee Tan, Heather Patisaul, Tom Zoeller, Joe Laasko, Joe DiGangi, Michael Lerner, Ken Cook, Linda Birnbaum

Expertise in Green Chemistry: Terry Collins

Expertise in Communications: Amy Kostant, Pete Myers, Carol Kwiatkowski, Tracy Woodruff, Karen Wang, Jeff Wise, Pat Hunt, Jane Muncke Ken Cook

NGO community/Scientific Societies: EHN, EWG, TEDX, CHE, Endocrine Society, SCN, PHRE, PEDS, Heal, Chemtrust, HEFN, community action groups, Mind the Store(safer chemicals: healthy families), Arlene Bloom, SEHN (Ted ~~Schecter~~Schettler), EDF, Non toxic Irvine (others ?) Mamavation

Expertise in Funding: Michael Lerner, Shorey Myers, Pete Myers, Linda Birnbaum, Jeff Wise

Representative from EDC Gordon Conference Planning (June 2-8 Les Diablerets Switzerland): Jodi Flaws

From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
To: "JPMyers@ehsic.org" <JPMyers@ehsic.org>
Sent: 4/15/2015 11:55:24 AM
Subject: Re: what do you think?

I havent read it but I dont see anything to get excited about. This has now been shown in a number of species heres one more.

From: Pete Myers <jpmyers@ehsic.org>
Date: Wednesday, April 15, 2015 at 4:50 AM
To: "Hunt, Patricia Ann" <pathunt@wsu.edu>, Fred Vom Saal <vomsaalF@missouri.edu>, Laura Vandenberg <lvandenberg@schoolph.umass.edu>
Subject: what do you think?

Bisphenol A Exposure during Oocyte Maturation in vitro Results in Spindle Abnormalities and Chromosome Misalignment in Bos taurus.

Ferris J, et al. Cytogenet Genome Res. 2015.
Show full citation

Abstract
Bisphenol A (BPA) exposure in humans is widespread, and BPA has been detected in a variety of samples including follicular fluid. BPA levels have been found to negatively correlate with the developmental potential of oocytes in women undergoing in vitro fertilization and to induce meiotic abnormalities experimentally in human and mouse models. BPA may detrimentally affect oocyte maturation, and different concentrations of exposure can cause various outcomes. Because of the importance of oocyte maturation on developmental potential, disturbances during this time can significantly impact oocyte viability. Here, bovine oocytes were matured in vitro with and without BPA treatment of the media. The levels of BPA taken up by the oocytes were much lower than the initial exposure. Medium treatment with 30 ng/ml resulted in an average of 2.48 ng/ml BPA measured in mature oocytes. These oocytes exhibited decreased maturation and increased incidence of spindle abnormalities. Only 57.4% of oocytes exposed to 30 ng/ml BPA reached maturity compared to 72.4% of controls ($p < 0.05$). Mature oocytes following BPA exposure displayed increased abnormal spindle morphology (67.9%) and chromosome dispersal (60%) compared to all other groups analyzed ($p < 0.05$). Thus, exposure to BPA during in vitro oocyte maturation has the potential to decrease oocyte quality. 2015 S. Karger AG, Basel.

Pete Myers, from a mobile phone

From: pathunt@vetmed.wsu.edu
To: Pete Myers <jpmyers@ehsic.org>
Sent: 6/1/2017 1:05:31 PM
Subject: Re: wrap your head around this!

Hi Pete-

Okay, both Terry's paper and your packaging plan are exciting. I'm sorry I missed the call - I got some horrible flu. Today I missed your call because I am at a retreat. We still haven't heard about the paper but should hear any day. I will let you know as soon as I do. In the meantime, I will be back in the office on Monday and we can talk if you have time.

Pat

Sent from my iPad

On Jun 1, 2017, at 9:07 AM, Pete Myers <jpmyers@ehsic.org> wrote:

> especially the mini-review of the occurrences of BPA (pp 3-7)
>
> Terry expects this to be accepted today and gave me permission to share it with you.
>
> Im thinking that I could give this and your forthcoming paper to Kristof once yours is accepted. The combination of the ubiquity of exposure (Collins) plus the extreme adversity of additive transgenerational impacts (you) could shake things up. Maybe not in the US. But I now have a 2 step access to the new President of France (who mentioned the need for stronger EDC regulations in the last debate with La Pen).
>
> What do you think?
>
> <2017-0601 BPA Green Chemistry Final-tjc.pdf>

From: Pete Myers <jpmyers@ehsic.org>
To: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
Sent: 5/15/2015 6:08:55 AM
Subject: Re: WSU

Thanks. Too bad! I don't know when Margaret will be on the west coast again, but it will happen.

> On May 14, 2015, at 6:30 PM, Hunt, Pat <pathunt@vetmed.wsu.edu> wrote:
>
> Hi Pete-
>
> Sorry it has taken me so long to respond. I am in Bend, OR with my family
> and things are a bit crazy. I saw your message last night but didnąt
> respond immediately because Iąd love to host you and Margaret and wanted
> to think about a way to work this in. Unfortunately, the timing is awful.
> The first 3 weeks of June are back-to-back and belly-to-belly for us.
> July, of course, is free and clear!
>
> Yours with regret,
>
> Pat
>
>
>
>
> On 5/13/15, 10:01 AM, "Pete Myers" <jpmyers@ehsic.org> wrote:
>
>> Margaret and I are going to be in the PNW to meet with Bruce Lanphear in
>> Vancouver. We could schedule a day or two at WSU 10-12 June if you were
>> going to be around. Might that be possible?
>> Best,
>> Pete
>

From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
To: Pete Myers <jpmyers@ehsic.org>
Sent: 5/14/2015 3:30:31 PM
Subject: Re: WSU

Hi Pete-

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Pat

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>Vancouver. We could schedule a day or two at WSU 10-12 June if you were
>going to be around. Might that be possible?
>Best,
>Pete

From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
To: "JPMyers@ehsic.org" <JPMyers@ehsic.org>
Sent: 8/15/2014 5:35:18 AM
Subject: Re: you ok with the quat story?

Yes, Lindsey did a really nice job. One of my colleagues at the meeting was sent the link by his vet staff, so it's already hitting the right people.

Sent from my iPhone

> On Aug 14, 2014, at 5:42 PM, "JPMyers@ehsic.org" <JPMyers@ehsic.org> wrote:
>
>
> best,
> p
>

From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
To: Amy Kostant <amy@sciencecom.org>, <thrubec@vt.edu>
Sent: 8/2/2017 8:57:09 AM
Subject: Re: your study featured on EWG's FB page

Thanks, Amy! I had not seen it, but this is great coverage.

From: Amy Kostant <amy@sciencecom.org>
Date: Wednesday, August 2, 2017 at 8:53 AM
To: patricia hunt <pathunt@vetmed.wsu.edu>, "thrubec@vt.edu" <thrubec@vt.edu>
Cc: Emily Copeland <emily@sciencecom.org>
Subject: your study featured on EWG's FB page

Hi Both,
Not sure if youve seen this yet: http://www.ewg.org/enviroblog/2017/07/disinfectant-mix-cleaning-products-linked-birth-defects-lab-animals#.WYFKFoTyv3g
LOTS of readers!
Congratulations,
Amy

Amy Kostant
Science Communication Network (SCN)
0: 301-654-6665
C: 202-255-6665
amy@sciencecom.org

From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
To: Amy Kostant <amy@sciencecom.org>
Sent: 6/9/2017 9:14:15 AM
Subject: Re: your upcoming paper

Attach: [Successive Generations of Exposure[1].docx]
Hi Amy-

We received word from the journal yesterday. They want us to add a paragraph to the discussion, which we are doing today. It should be accepted early next week. Since it is a PLoS journal, it is open access and will be published almost immediately. I will let you know as soon as I receive the formal acceptance. In the meantime, I have attached our penultimate draft it is NOT the final, nor does it include the figures, but the abstract, intro and discussion will be useful to you, I think.

Thanks,

Pat

From: Amy Kostant <amy@sciencecom.org>
Date: Friday, June 9, 2017 at 5:27 AM
To: patricia hunt <pathunt@vetmed.wsu.edu>
Cc: Emily Copeland <emily@sciencecom.org>
Subject: your upcoming paper

Hi Pat,
Im looking forward to seeing you on the 23.
As you know, Petes excited about your upcoming paper on transgenerational exposures to BPA. His excitements contagious, so if possible, may I see the paper (not for distribution)? Do you have a publication date in mind?
Thanks.
Amy

Amy Kostant
Science Communication Network (SCN)
0: 301-654-6665
C: 202-255-6665
amy@sciencecom.org

Germline and reproductive tract effects intensify in male mice with successive generations of estrogenic exposure

Tegan S. Horan, Alyssa Marre, Terry Hassold, Crystal Lawson, and Patricia A. Hunt*

School of Molecular Biosciences, Center for Reproductive Biology, Washington State University, Pullman, Washington, United States of America

*Corresponding author:
E-mail: pathunt@vetmed.wsu.edu (PAH)

Abstract

The hypothesis that developmental estrogenic exposure induces a constellation of male reproductive tract abnormalities is supported by experimental and human evidence. Experimental data also suggest that some induced effects persist in descendants of exposed males. These multi- and transgenerational effects are assumed to result from epigenetic changes to the germline, but few studies have directly analyzed germ cells. Typically, studies of transgenerational effects have involved exposing one generation and monitoring effects in subsequent unexposed generations. This approach, however, has limited human relevance, since both the number and volume of estrogenic contaminants has increased steadily over time, intensifying rather than reducing or eliminating exposure. Using an outbred CD-1 mouse model, and a sensitive and quantitative marker of germline development, meiotic recombination, we tested the effect of successive generations of exposure on the testis. We targeted the germline during a narrow, perinatal window using oral exposure to the synthetic estrogen, ethinyl estradiol. A complex three generation exposure protocol allowed us to compare the effects of individual, paternal, and grandpaternal (ancestral) exposure. Our data indicate that multiple generations of exposure not only exacerbate germ cell exposure effects, but also increase the incidence and severity of reproductive tract abnormalities. Taken together, our data suggest that male sensitivity to environmental estrogens is increased by successive generations of exposure.

Author Summary

Developmental exposure to manmade chemicals that interfere with endogenous hormones (endocrine disrupting chemicals) has been reported to adversely affect male reproductive health, increasing the incidence of reproductive tract abnormalities and reducing sperm production. Experimental evidence suggests that some exposure effects can persist in unexposed descendant males. To date, however, studies of these transgenerational effects have failed to accurately model human exposure, which spans multiple generations and

involves an increasing number and diversity of endocrine disrupting chemicals. Using a quantitative measure of exposure effects on the germline, we assessed the effects of successive generations of estrogenic exposure in mice. We found that multiple generations of exposure not only exacerbated previously reported effects on the male germline, but elicited reproductive tract defects that increased in frequency and severity. These results have important implications for human reproductive health, suggesting that multiple generations of exposure to common endocrine disrupting chemicals may increase male sensitivity to exposure.

Introduction

Data from human populations around the world provide evidence of a marked decline in male fertility during the past several decades. For example, a comprehensive analysis in 2000 of data from more than 100 studies in Western countries provided evidence of a decline in human spermatogenesis during the preceding 50 years [1]. More recent longitudinal cross-sectional studies suggest reductions in both sperm count and quality among young men (ages 18-37) in China (2001-2015;[2]), Spain (2001-2011;[3]), France (1989-2005;[4]), Denmark (1996-2010;[5]), and Finland (1998-2006; [6]). Changes in sperm production have coincided with increases in the incidence of other reproductive defects, including hypospadias, cryptorchidism, and testicular germ cell cancers (reviewed in [7]), and the combined spectrum of reproductive effects has been termed testicular dysgenesis syndrome (TDS; [8]). The observed changes correspond to the rapid introduction of manmade chemicals in the postwar era, and were originally hypothesized to result from exposure to maternally- or environmentally-derived estrogens [9]. Subsequent experimental data, however, have provided evidence that male reproductive abnormalities can be induced by developmental exposure to different types of endocrine disrupting chemicals (EDCs; reviewed in [10,11]). Given the rapid increase in the variety and ubiquity of EDCs in our environment and the adverse reproductive effects ascribed to some of these chemicals, the implications for humans are significant.

The most compelling evidence of an effect of developmental estrogenic exposure on human male reproductive health comes from studies of diethylstilbestrol (DES) exposed sons. From the 1940s through the 1970s, DES was prescribed to millions of pregnant women to prevent miscarriage. This treatment not only was not efficacious, but increased the incidence of a variety of reproductive disorders, including cancers in both male and female offspring (reviewed in [12]). Although DES daughters have been studied more extensively, in DES sons and in male mice exposed prenatally to DES, the incidence of cryptorchidism, underdeveloped testes, and testicular cancer is increased, and sperm count and quality is decreased [13–16]. Further, although the lack of information on sources, levels and timing of exposure precludes systematic studies of other developmental estrogenic exposures in humans, epidemiological studies suggest etiological links between environmental exposures and changes in spermatogenesis and the incidence of testicular germ cell cancers of fetal origin (reviewed in [7,17]).

Evidence that the effects of exposure may be transmitted to subsequent, unexposed generations is accumulating. Because exposure not only can directly affect the exposed individual (F0), but also his or her germline, effects evident in generations derived from this germline (the F1 in the case of male exposure, but both the F1 and F2 generations in the case of fetal exposure involving the female) are said to be multigenerational. For an effect to be considered transgenerational, it must be evident in the first unexposed generation (F2 and F3 for male and female exposures, respectively). Transgenerational effects in mammals – presumably resulting from epigenetic changes to the germline – have been reported in numerous studies (e.g., [18–25]). Few studies, however, have focused on germ cells [26–28], and the evidence supporting the persistence and transmission of specific germline alterations remains insufficient to convince some skeptics (e.g. [29–31]). Direct effects on the developing male germline have been induced by perinatal exposure to exogenous estrogens in mice and rats, with adverse effects reported on both gonocyte number and adult sperm production [32–

36]. In addition, we previously demonstrated an effect on the developing spermatogonial stem cell (SSC) induced by brief postnatal exposure coinciding with the formation of the SSC lineage in male mice and evident as a reduction in meiotic recombination levels in descendant spermatocytes [37].

Documenting transgenerational effects in humans is challenging. Assessing potential transgenerational transmission of DES-induced effects will require analysis of an additional generation of descendants and, for most common environmental chemical contaminants, assessment likely will never be possible due to the nature of human exposure: Typically, humans are not exposed for only a single generation. Instead, exposures persist over time or become more diverse as new chemical variants are introduced.

To our knowledge, the effects of successive generations of exposure on male reproduction have not been addressed. Thus, we decided to use a sensitive, quantitative measurement of exposure, meiotic recombination, to assess the effect of exposures spanning multiple generations. We utilized an outbred mouse model and a complex three-generation scheme (Fig 1A) involving low-dose, neonatal exposure to the synthetic estrogen, ethinyl estradiol. Our data not only demonstrate an increase in the severity of exposure-induced effects on meiotic recombination with successive generations of exposure, but also an unexpected increase in both the incidence and severity of male reproductive tract aberrations. Taken together, our findings suggest that continued exposure spanning several generations will have cumulative effects on male reproductive health.

Results

We recently reported that neonatal estrogenic exposure induces permanent meiotic effects in adult outbred CD-1 and inbred C3H, but not C57BL/6J male mice [37]. Germ cell transplantation experiments demonstrated that the meiotic phenotype was due to alterations in

the spermatogonial stem cells (SSCs) of the testis, a lineage thought to be determined during the window of exposure used in the study [38–40]. The SSC is many cell divisions upstream of meiotic entry; thus, rather than affecting the meiotic DNA double strand break (DSB) repair process per se, it is likely that changes induced in the SSC population altered the recombination set point. Consistent with this, we found no difference in DSB formation or synaptonemal complex length in exposed and control males [37]. Studies to determine how exposure alters the SSC epigenome are in progress, and ultimately will provide important insight to recombination control in male mammals. In the interim, because recombination provides a quantitative measure of an exposure effect on the germline, it provides a direct means of tracing effects through generations to determine if they are multi- or transgenerational.

Our previous studies demonstrated that exposure to either bisphenol A (BPA) or ethinyl estradiol from 1-12 days postpartum (dpp) significantly reduced meiotic recombination (as assessed by the number of foci of the DNA mismatch repair protein, MLH1 in pachytene spermatocytes) in adult males. Daily oral doses of 0.25 ng/g ethinyl estradiol (roughly equivalent to a daily oral contraceptive dose) exerted the strongest effect, causing a 5% reduction in MLH1 values in inbred C3H males [37]. Although this difference appears subtle, the direct biological consequence is the elimination of spermatocytes. Cells with one or more pairs of homologous chromosomes that fail to form a crossover site will not yield sperm, because the presence of unpaired chromosomes at the first meiotic division triggers checkpoint-induced spermatocyte elimination [41,42].

We were interested not only in analyzing second- and third-generation descendants of exposed males for the transgenerational persistence of meiotic effects, but also in assessing the effects of successive generations of exposure. Accordingly, we developed the three-generation exposure protocol outlined in Fig 1 and S1 Fig, and conducted all analyses on 12-week-old adult males. To track both generational and individual exposure history, F0 founder exposed males were designated as ‘E’, and ‘E’s and ‘0’s used to designate exposure or placebo

treatment, respectively in subsequent generations (Fig 1B, C). For example, EE males represent F1 generation exposed sons with two generations of exposure; E00 males, F2 grandsons two generations removed from the founder exposure; and EEE males, F2 grandsons with three successive generations of exposure. In this paradigm, E0 and E00 males serve as important negative controls for EE and EEE exposure groups.

To eliminate genetic variability, we initiated our three-generation studies using inbred C3H males; however, four of the nine founder males proved infertile with orchitis. When we attempted the study using inbred 129 males orchitis was not observed, but only one of four exposed males proved fertile. We next turned to outbred CD-1 males.

Although the use of outbred animals introduces genetic variability, our previous studies demonstrated meiotic effects in neonatally exposed CD-1 males (i.e., an average decrease in adult males of 1.3 MLH1 foci for BPA and 2.5 for ethinyl estradiol) and suggested that exposed CD-1 males are fertile [37]. The highly significant difference between ethinyl estradiol and placebo exposed males suggested that, despite genetic variation, we would be able to discern generational differences using CD-1 males, thus, we conducted our studies on this outbred background.

Exposure-induced reproductive tract abnormalities are additive

Although our initial focus was on exposure-induced meiotic recombination effects, we observed unexpected malformations of the vas deferens in two of the three CD-1 founder males. Accordingly, we evaluated the vas deferens of all descendant males and noted an increase in both the incidence and severity of defects with subsequent generations of exposure. Specifically, in addition to the abnormal kinking of the vas deferens observed in two F0 founders, an even more severe aberration that we termed ‘collapsed’ emerged in EE F1 males

and increased in incidence in EEE F2 males (Fig 2). In addition, a new and severe phenotype, testis fibrosis, emerged in the third generation.

To assess the effect of successive generations of exposure on the incidence of vas deferens malformations (i.e. the proportion of males with either kinked or collapsed phenotypes), we compared F1 and F2 males with multiple generations of exposure (EE and EEE) to those with only one (E0 and E00). Among F1 males, the incidence of abnormalities was significantly increased in EE by comparison with E0 males (48.0% (12/25) vs. 10.0% (3/30), respectively; $X^2 = 8.1$, $p < 0.01$; Fig 2D). A similar comparison of F2 males (EEE and E00) demonstrated an even stronger exposure effect (90.0% (36/40) vs. 11.4% (4/35), respectively; $X^2 = 43.2$, $p < 0.0001$; Fig 2D). As a further test of the effect of successive generations of exposure, we compared F1 (EE) and F2 (EEE) males and found a significant increase in the incidence of defects in F2 males ($X^2 = 12.0$, $p < 0.001$; Fig 2D).

The severity of reproductive tract abnormalities also increased with successive generations of exposure (Fig 2D, E). In the F1 generation, 28.0% (7/25) of EE but none (0/30) of the E0 males exhibited the more severe collapsed vas deferens phenotype (Fig 2D). In the F2 generation, the incidence of this severe abnormality was 47.5% (19/40) in EEE, but only 5.7% (2/35) in E00 grandsons (Fig 2D). Importantly, although the collapsed phenotype was evident in all three families, it was most pronounced in families 1 and 3 (Fig 2E, S1 Fig and S2 Fig). Notably, the family with the lowest incidence of the collapsed phenotype, family 2, was derived from the only founder with a normal vas deferens. Family 1 appeared most affected and, by comparison with family 2, the collapsed phenotype occurred in 50.0% (4/8) vs.11.1% (1/9) of EE males (not significant), and 88.9% (8/9) vs. 25.0% (4/16) of EEE grandsons, respectively ($X^2 = 7.0$, $p < 0.01$; Fig 2E). Intriguingly, in family 1 two E00 grandsons (18.2%) also exhibited the collapsed phenotype, providing the only examples of the severe phenotype among E00 males.

Unexpected abnormalities were not confined to the ductal system, as a new, severe testis phenotype emerged in the third generation. Fibrotic testes, frequently characterized by fusion of the testis and reproductive tract (Fig 3A) was observed in a minority of F2 generation males in each exposure family (Fig 2E). As shown in Fig 3B, histological analysis of testes from affected males revealed prominent cysts (not evident in this image), apparent expansion of interstitial tissue, and atrophied seminiferous tubules devoid of active spermatogenesis. The phenotype was confined to F2 males but included individuals receiving either two or three generations of exposure, and in 14/17 cases both testes were affected. Among EEE males, the frequency was similar across families, with 20.0% (2/10), 21.1% (4/19), and 21.1% (4/19) affected for families 1, 2, and 3, respectively (Fig 2E). Unlike the vas deferens phenotype, the fibrotic testis phenotype was not obviously related to paternal phenotype, indeed 16.3% (7/43) of F2 males with an interrupted generation of exposure (i.e. E0E, but not EE0) exhibited fibrosis (Fig 3C, S1 Fig).

Meiotic effects worsen with successive generations of exposure

In addition to eliciting more severe reproductive tract aberrations, multiple generations of estrogenic exposure exacerbated the meiotic recombination phenotype that was the original focus of our analysis. As in our previous studies [37], we analyzed recombination in pachytene stage spermatocytes by counting MLH1 foci in preparations immunostained with antibodies to both SYCP3 (a component of the synaptonemal complex or SC) and MLH1, a mismatch repair protein that localizes to the majority of meiotic crossovers [43]. In our previous studies, the MLH1 mean for placebo treated males was 24.6 ± 0.3 and both BPA and EE exposure induced a significant decrease (i.e., 1-2.5 foci, depending upon the exposure) [37]. Thus, the means of F0 founder males (23.7 ± 0.3, 22.7 ± 0.4, and 22.1 ± 0.3 for family 1, 2, and 3, respectively) fell within the expected range for exposed males. To compare recombination levels across

generations and among different categories of F1 and F2 males, mean MLH1 counts were derived by pooling cells from males of the same generation and exposure category.

To assess the effect of successive generations of exposure, we used one-way ANOVA to compare mean MLH1 counts in exposed F0 males with those in F1 and F2 males exposed for two or three generations (Fig 4A; $F = 29.4$, $p < 0.0001$). Significant differences between groups were determined by a Tukey-Kramer post-hoc test. By comparison with F0 founders (22.8 ± 0.2) we found a small but nonsignificant decrease in mean MLH1 counts in EE sons (22.7 ± 0.1), but a significant reduction in EEE grandsons (21.8 ± 0.1; $p < 0.05$). In addition, MLH1 means were lower in F1 EE (22.7 ± 0.1) than E0 sons (23.1 ± 0.1, $p < 0.05$). Similarly, the mean was significantly lower in EEE by comparison with E00 F2 males (21.8 ± 0.1 and 22.8 ± 0.1, respectively; $p < 0.05$).

Because family 1 exhibited the strongest effect, we assessed each family individually to determine if trends were consistent across families (Fig 4B-D). In family 1, MLH1 means were significantly lower in both F1 EE sons (21.8 ± 0.1) and F2 EEE grandsons (22.0 ± 0.2) by comparison with the founder mean (23.7 ± 0.3; $p < 0.05$; Fig 4B). For families 2 and 3, reductions were evident in F2 EEE males, but the differences were not statistically significant (Fig 4C, D). Thus, all families exhibited the same trend; differences among them prompted us to consider a paternal effect on recombination.

Recombination exhibits a strong paternal effect

As observed for vas deferens abnormalities, the recombination phenotype of offspring appeared to be influenced by paternal phenotype. Specifically, the extent to which the phenotype worsened with successive generations of exposure not only varied among families, but also among the offspring of males within a family, with a more pronounced effect in sons of males with higher mean MLH1 counts. For example, the founder of family 1 had the highest mean MLH1 level (23.7 ± 0.3), and his seven F1 sons (EE) all had lower mean values (ranging

from 21.0 ± 0.3 to 23.4 ± 0.3; Fig 4B, S3 Fig). In contrast, in the other two families where founder MLH1 means were lower (22.7 ± 0.4, and 22.1 ± 0.3 for family 2 and 3, respectively), means in F1 EE sons (23.2 ± 0.1 and 22.8 ± 0.2, respectively) were not significantly different from the F0 founder mean (Fig 4C, D).

A comparison of the F2 sons of F1 EE fathers provided further evidence of this paternal effect. For example, the two F1 EE males in family 2 that were mated to produce F2 EEE males had very different MLH1 means (25.1 ± 0.4 and 22.6 ± 0.3). Although the five F2 EEE offspring of each male had lower mean MLH1 counts than their fathers (23.0 ± 0.2, $t = 4.6$, $p < 0.0001$, and 21.4 ± 0.2, $t = 3.1$, $p < 0.01$ respectively; Fig 5), the means and ranges of the two groups of males were remarkably different. Importantly, the magnitude of the reduction was greater in F2 sons of the F1 male with the high MLH1 count. Similar paternal effects were observed among the offspring in all three families (S3 Fig); however, the impact of the paternal phenotype on the response to exposure was most pronounced in family 3, where the MLH1 mean of one F1 male was particularly low (20.7 ± 0.2). The mean for the F2 EEE sons of this male (20.1 ± 0.2) did not differ significantly from the F1 EE father, making this the only group of F2 EEE males that did not demonstrate an additional reduction in recombination levels by comparison with their father (Fig 5).

Recombination failure increases with successive generations of exposure

The variability induced by the use of outbred males, coupled with the male reproductive tract abnormalities we encountered, confounded the use of standard measurements of impaired male fertility. Thus, we elected to directly measure meiotic impairment by scoring cells with lethal defects, i.e., the frequency of pachytene stage cells containing one or more SCs lacking an MLH1 focus. As expected, exposure-induced reductions in meiotic recombination resulted in an increase in recombination failure (Fig 6A). A comparison of males exposed each generation

(i.e. E, EE, and EEE) showed a significant increase in the incidence of these cells over three successive generations. Specifically, recombination failure was observed in 10.8% (9/83) of cells from F0 founder males, 14.2% (91/643) of cells from F1 EE sons, and 34.9% (248/710) of cells from F2 EEE grandsons ($X^2 = 87.9$, $p < 0.0001$; Fig 6B). Although the difference in levels of recombination failure between founders and EE sons was not significant, levels in EEE grandsons were significantly higher by comparison with both founders ($X^2 = 18.6$, $p < 0.0001$) and EE fathers ($X^2 = 76.5$, $p < 0.0001$). This trend held among father-son comparisons within individual families (S4 Fig).

The recombination failure phenotype also provided a means of assessing the transgenerational persistence of meiotic effects. A comparison of levels in E, E0, and E00 males not only did not reveal a decrease in recombination failure levels in subsequent unexposed generations but provided evidence of a slight increase across generations ($X^2 = 11.2$, $p < 0.01$; Fig 6B). However, the effect was only statistically significant in the pooled data and a definitive trend was not observed across all families (S4 Fig). Thus, although these data are consistent with transgenerational persistence of the phenotype, clearly additional analyses are warranted.

Both ancestral and individual exposure influences the male recombination phenotype

As detailed above, our data from exposure families provide evidence of increases in both the severity of reproductive tract aberrations and meiotic effects with successive generations of exposure. Our breeding scheme generated three different types of two-generation exposure males (F1 EE males, and F2 E0E and EE0 males), and these males provide a means of separating ‘ancestral’ and direct or ‘individual’ exposure effects. To assess the effect of individual exposure, we compared EE0 and E0E F2 males. Ancestral exposure is common to both, but only E0E males received direct exposure as neonates. A comparison of

pooled data for these F2 males suggests stronger effects as a result of individual exposure. Vas deferens defects were more common in E0E males, with abnormal phenotypes in 87.2% (34/39) males vs. 15.4% (4/26) in the EE0 males ($X^2 = 30.2$, $p < 0.0001$; S5 Fig A). These defects were also more severe in E0E males, with collapsed phenotypes in 38.5% (15/39) of males vs. 3.8% (1/26) of EE0 males ($X^2 = 8.3$, $p < 0.01$; S5 Fig A). Similarly, a stronger reduction in MLH1 counts was evident in E0E males, with means of 22.1 ± 0.07 for E0E and 22.8 ± 0.10 for EE0 (Tukey-Kramer post-hoc, $p < 0.05$; S5 Fig B). Further, the proportion of cells with recombination failure was also higher: 26.4% (198/749 cells) for E0E vs 18.2% (115/633 cells) for EE0 ($X^2 = 12.9$, $p < 0.001$; S5 Fig C). These trends held when we examined individual families with one exception: in family 3, E0E males showed a lower, although not significant, decrease in recombination failure by comparison with EE0 males (16.6% (42/253) and 20.1% (56/279), respectively; S6 Fig).

Discussion

Growing evidence suggests that developmental exposure to EDCs may exert effects that span multiple generations (e.g., [19,21,22,24,25,44–48]), but little attention has focused on an equally important question with obvious human relevance - the effect of successive generations of exposure. In this respect, our study is unique since, rather than exposing one generation and assessing the persistence of effects in subsequent generations, we exposed multiple generations and compared the severity of effects across generations. Further, in contrast to trans- and multigenerational studies that have used pooled data to assess effects in each generation, we assessed differences among individual families, focusing on relationships between exposure effects evident in fathers, sons, and grandsons.

Most reports of transgenerational effects have utilized *in utero* exposures that can disrupt fetal development as well as epigenetic reprogramming of the germline. Instead, we attempted to target the developing germline by exposing male mice postnatally during the period thought to coincide with the establishment of the spermatogonial stem cell (SSC) pool [49]. Effects elicited in the male reproductive tract demonstrate that our exposure also affected somatic differentiation of the male reproductive tract but, importantly, these unexpected effects lend further support to the conclusions of our meiotic studies.

The major finding from our study is that, in male mice, estrogenic exposure spanning several generations exacerbates reproductive abnormalities induced by exposure. Our exposure paradigm (Fig 1) allowed us to identify both ancestral and paternal effects on the incidence and intensity of exposure phenotypes. First, ancestral exposure influenced the magnitude of the effect, since exposed grandsons – even those sired by nonexposed fathers - exhibited the most severe reproductive aberrations. Second, paternal phenotype strongly affected the magnitude of the meiotic effect in exposed offspring. Taken together, our data add a concerning new dimension to the estrogen hypothesis [8]. Specifically, our findings suggest that neonatal estrogenic exposure can affect both the reproductive tract and sperm production in exposed males, and exposure effects are exacerbated by exposure spanning multiple generations. Because estrogenic chemicals have become both increasingly common and ubiquitous environmental contaminants in developed countries, the implications for humans are serious. Indeed, it is possible effects are already apparent, with population-based studies from the U.S., Europe, Japan, and China reporting reductions in sperm counts/quality [2,3,5,6,50–53] and male fertility (reviewed in [7]) within a span of several decades.

Successive generations of estrogenic exposure exacerbate male reproductive effects

We identified three phenotypes that increased in severity with successive generations of neonatal estrogenic exposure. Two of the effects, malformations of the vas deferens and altered levels of meiotic recombination, were evident in first generation exposure males (F0) but were exacerbated by successive generations of exposure. The third and most severe phenotype, fibrotic testes, was observed only in third-generation (F2) males.

The vas deferens, which is normally a straight tubule, exhibited kinking along its entire length in two of the three founder males. This phenotype not only was more pronounced in exposed descendants, but became markedly more severe (i.e., 'collapsed') after two generations of successive exposure (28% of EE males; Fig 2) and was the predominant phenotype in EEE males (47.5%; Fig 2).

Altered Hox gene expression is known to affect the developing male reproductive tract, and similar abnormalities of the vas deferens - described as partial homeotic transformation of the vas deferens into an epididymis - have been reported in *Hoxa10* and *Hoxa11* mutant adults [54–56]. In our studies, however, and in postnatal DES exposure studies where similar vas deferens effects were observed [57–59], fetal development of the male reproductive tract would be unaffected. Thus, the abnormalities induced by postnatal exposure must result from delayed or impaired differentiation. Because the proximal-to-distal coiling of the epididymis concludes around the time of birth [60] our findings, in conjunction with those from DES exposed males, suggest that neonatal exposure to estrogens prevents cessation of coiling in the male reproductive tract. A critical role of androgens in the perinatal elongation and coiling of the differentiating Wolffian ducts/epididymis has been suggested previously [60,61], and DES-induced reproductive tract defects have been suggested to result from disruption of the neonatal estrogen-androgen balance [58,59]. Our data, however, add a new level of complexity regarding sensitivity to exposure: Although ethinyl estradiol and DES have similar IC50 and relative binding affinities for estrogen receptors [62], the effects we observed among EEE grandsons

were more severe than with DES-induced abnormalities [57–59] despite the fact that the level of estrogenic exposure was markedly lower (18.3 ng ethinyl estradiol vs. 60 μg DES). Thus, our data suggest that sensitivity to exposure is increased with successive generations of exposure.

The emergence of a new phenotype, fibrotic testes exhibiting complete spermatogenic failure, in the F2 generation further underscores the cumulative consequences of multiple generations of exposure. Because this phenotype only emerged in the last generation and was not evident until the males were killed for analysis, we can only speculate on the genesis of this abnormality. However, given the high frequency of orchitis encountered when we attempted to initiate studies using inbred C3H males, it seems likely that exposure may interfere with the formation of the blood-testis barrier. In addition to being common (20.8% and 16.3% of EEE and E0E males, respectively; Fig 3C), the fibrotic phenotype is particularly interesting because a similar phenotype has been reported in CD-1 males following prenatal exposure to high doses (100 ng/g) of DES [63]. Because we utilized a considerably lower dose of ethinyl estradiol (0.25 ng/g), the appearance of this phenotype in third-generation males supports the hypothesis that exposure sensitivity is heightened by multiple generations of exposure.

The phenotype that was our original focus of study, reduced levels of meiotic recombination in exposed males, also was exacerbated by successive generations of exposure. Recombination levels have been well characterized in mice [64–66], and changes in MLH1 counts provide a sensitive and quantitative means of assessing the effects of estrogenic exposure [37]. Mean MLH1 counts in three generation exposure lineage F2 males (EEE) were not only significantly lower than levels in their grandfathers (F0 founder males), but also by comparison with single generation exposure F2 males (E00). Although the recombination changes may appear subtle, from the standpoint of successful spermatogenesis, they are substantial. The mouse genome consists of twenty pairs of chromosomes, and a physical connection (chiasma), between each pair is essential for normal segregation of homologous

chromosomes at the first meiotic division. Because chiasmata are established at sites of meiotic recombination, an exposure-induced reduction in MLH1 counts should increase the incidence of SCs lacking an MLH1 focus, an expectation borne out in our previous studies [37]. Our current results not only confirm this finding, but demonstrate a worsening of the effect with increasing generations of exposure. Indeed, the 35% incidence of recombination failure observed in EEE grandsons (Fig 6B) is particularly concerning. Recombination failure increases the incidence of univalents at metaphase I, and these cells are effectively eliminated from the spermatocyte pool through the robust actions of the spindle assembly checkpoint (SAC; [41,42]), a finding confirmed in our initial studies [37]. Thus, the net effect of recombination changes induced by successive generations of estrogenic exposure would be reduced levels of sperm production. Because sperm counts are variable among outbred males, and the high incidence of reproductive tract abnormalities would confound simple fertility measurements, we did not attempt to quantitate the exposure effect on male fertility. Based on recombination failure levels observed in F0 founders (10.8%) and F1 EE males (14.2%), however, we suspect that the effects of exposure in the first two generations are too subtle to elicit significant effects on fertility. In contrast, although we did not breed F2 males the high incidence of testis fibrosis in this generation suggests a marked increase in infertility with successive generations of exposure.

Paternal phenotype affects the severity of exposure effects in descendants

The incidence and severity of meiotic and reproductive tract defects varied among the three exposure families in our study. CD-1 is an outbred strain and, to obtain successive generations, males were mated with unexposed females. Thus, phenotypic variation is expected. The variability among males, however, allowed us to discern an effect of paternal phenotype on the severity of meiotic effects in subsequent generations. Specifically, a larger

reduction in mean MLH1 counts was evident among exposed sons of fathers with high recombination levels by comparison with sons of fathers with very low recombination levels (Fig 5). In essence, our data raise the possibility of a maximal effect of estrogenic exposure on meiotic recombination that, once reached, cannot be further exacerbated. Thus, in future studies it will be important to assess effects of exposure spanning more than three generations.

Ancestral and individual exposures exacerbate reproductive defects

Our data also provide evidence of the persistence of ancestral exposure effects. Cumulative effects of multiple generations of exposure provide strong evidence that a combination of both ancestral and individual exposures elicits the most pronounced effects, as F2 males with both (e.g., E0E grandsons) exhibited more frequent and severe phenotypes than did males without individual exposure (EE0 grandsons). Further, it is notable that testicular fibrosis was observed only in males with both grandpaternal and individual exposures as neonates (i.e., E0E and EEE males), providing further evidence that the combination of an ancestral and individual exposure was both necessary and sufficient to elicit the most severe reproductive effects.

Summary and implications for human health

Increasing evidence that developmental exposure to endocrine disrupting chemicals can elicit phenotypic effects that are transgenerationally inherited has sparked interest in exposure-induced epigenetic changes (reviewed in [67,68]). Associated changes in DNA methylation [69], histone modifications [70], and small RNAs [47,71] have been reported, but causative links between these alterations and transgenerational disease phenotypes are equivocal. Few studies have focused on changes to the germline (e.g., [26,28]) or traced the transmission of

specific epimutations across generations (e.g., [27,70]). The complex multigenerational exposure paradigm that we used allowed for the detection of exposure effects directly in germ cells and made it possible to quantitatively compare effects in descendant males. Thus, in addition to assessing trans- and multigenerational effects, our study represents a logical and important next step in exposure studies - assessing the effects of multiple generations of exposure. Because both exposure-induced meiotic and reproductive tract defects increased in frequency and severity with successive generations of exposure, our data provide evidence that persistent exposure increases male reproductive tract sensitivity. Given the dramatic increase in both the number and complexity of environmental chemical contaminants during the past several decades, our findings have obvious human relevance. Indeed, evidence of global reductions in sperm production [1,3,6,50–53,72] suggest that similar effects may already be manifest in human populations, and underscore the importance of understanding the levels and types of exposures in human populations.

Materials and Methods

Animals

Outbred CD-1 mice (Harlan Laboratories) were housed in polysulfone cages on ventilated racks (Allentown Inc., Jag 57 micro isolator model) in a pathogen-free facility. Cages contained Sanichip 7090A bedding (Harlan Laboratories) and a nestlet (Ancare) for enrichment. Drinking water and food (Purina Lab Diet, 5K52) were provided ad libitum.

All experiments were approved by the International Animal Care and Use Committee (IACUC) at Washington State University, which is fully accredited by the American Association for Accreditation of Laboratory Animal Care.

Exposures

All males were treated from 1-12 days postpartum (dpp) with either 0.25 ng/g/day ethinyl estradiol (Sigma-Aldrich, E4876) or equal volume ethanol/corn oil placebo. Ethinyl estradiol was dissolved in 100% ethanol and diluted in tocopherol-stripped corn oil (MP Biomedicals) and administered orally by pipette. Doses were calculated based on mean pup weight (g) for this strain. The 0.25 ng/g ethinyl estradiol dose was chosen because it was used as a positive control in our previous studies and elicited a strong meiotic effect [37]. The dose used is roughly equivalent to that in contraceptive pills (15 – 30 µg).

Three estrogen-exposed males served as the F0 founders of three exposure families. At 6 wks. of age, these founder males were paired with unrelated CD-1 females to produce second-generation (F1) males. Each founder produced four litters; two were treated with ethinyl estradiol and two with placebo. At sexual maturation, one randomly-selected male from each litter was mated to produce four litters of third-generation (F2) males, two treated with ethinyl estradiol and two placebo-treated. This paradigm yielded two groups of F1 males with either one (E0) or two (EE) generations of exposure (n = 30 and 25 mice, respectively), and four groups of F2 males having: 1) a single ancestral exposure (E00; n = 35); 2) an ancestral and paternal exposure (EE0; n = 27); 3) an ancestral and individual exposure (E0E; n = 43); or 4) three successive generations of exposure (EEE; n = 48; Fig 1). Males of all generations were killed at 12 wks. of age and their testes and reproductive tracts removed for analysis. Similarly, males from unexposed lineages (n = 27) were treated with placebo from 1-12 dpp, killed at 12 wks. of age, and their testes and reproductive tracts were analyzed as a control group.

Reproductive tract analysis

Vas deferens and epididymides were dissected and images captured using a Leica DFC295 camera on a Leica dissection microscope. The morphology of the vas deferens was assigned a numerical score of 1 (normal), 2 (kinked), or 3 (collapsed) by three independent observers who were blinded with respect to exposure status.

Immunohistochemistry

Testes exhibiting signs of fibrosis were fixed in Bouins solution, embedded in paraffin, and sectioned. Sections were deparaffinized, rehydrated, and stained with hematoxylin.

Spermatocyte preparations and immunostaining

Spermatocyte preparations were made according to the method developed by Peters [73]. Slides were incubated overnight in a humid chamber and washed with 0.4% Photo-flo 200 solution (Kodak Professional). Immunofluorescence staining of slides was performed as described previously [37]. Slides were simultaneously stained with MLH1 primary antibody (Calbiochem, PC56, at 1:60) and SYCP3 primary antibody (Santa Cruz biotechnology, sc-74569, at 1:300), and counterstained with Alexa Fluor 488-conjugated AffiniPure Donkey Anti-Rabbit (AFDAR) secondary antibody (Jackson Immunoresearch Laboratories, Inc., 711-545-152, at 1:60) and Cy3-conjuagted AffiniPure Donkey Anti-Mouse (CDAM) secondary antibody (Jackson Immunoresearch Laboratories, Inc., 715-165-150, at 1:1000).

MLH1 analysis

Cells were imaged using a Zeiss Axio Imager epifluorescence microscope. MLH1-FITC, SYCP3-TRITC, and DAPI were imaged sequentially, adjusted using Zeiss Axiovision software, and the number of MLH1 foci in composite images of MLH1 and SYCP3 counted by two independent observers who were blinded with regard to exposure status. 25-30 pachytene spermatocytes were scored per animal; minor counting discrepancies were resolved but cells with major scoring discrepancies, poor staining, or synaptic defects were excluded.

Statistical analyses

Among-group differences in mean MLH1 foci counts were analyzed by one-way ANOVA. For statistically significant differences ($p < 0.05$), a Tukey-Kramer post-hoc test was performed to infer which groups differed. Comparisons of mean MLH1 foci counts between F1 fathers and their F2 sons were analyzed by one-tailed t-test. Chi-square analyses were used to determine significance in the proportion of vas deferens aberrations and of cells containing SCs lacking an MLH1 focus.

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Figure Legends

Fig 1 Multigenerational exposure paradigm. (A) F0 males (designated E) were treated from 1-12 dpp with 0.25 ng/g ethinyl estradiol and bred with unexposed females to produce F1 males that received daily oral doses of either ethinyl estradiol (EE; red) or placebo (E0; blue) from 1-12 dpp. Representative F1 males chosen at random were bred with unexposed females to generate F2 generation males that, like the F1, were either exposed (E0E and EEE; red) or placebo treated (E00 and EE0; blue). After mating, all males were killed at 12 weeks of age for reproductive tract and testis analysis (B) Summary of abbreviations and specific exposure(s) represented by each. (C) Summary of animal numbers in each treatment group.

Fig 2 Successive generations of estrogenic exposure increase both incidence and severity of vas deferens malformations. (A-C) Epididymis and attached vas deferens showing normal (A), 'kinked,' characterized by a convolution along the length of the vas deferens (B), and 'collapsed', characterized by curling of the 'kinked' duct on itself (C) phenotypes. (D) Comparison of the frequency of kinked (light blue) or collapsed (dark blue) phenotypes in placebo controls (n = 27), the 3 F0 founders, 30 E0 and 25 EE F1 sons, and 35 E00 and 40 EEE F2 grandsons. Incidence of abnormal phenotypes was significantly higher after successive generations of exposure: Asterisks denote level of significance of comparisons (see text for details). (E) Pedigrees of exposed families with vas deferens phenotype denoted by square color: normal (white), kinked (light blue), and collapsed (dark blue), and black denoting fibrotic testes. Blue lines of descent indicate placebo treated lineages, red denote estrogen treated. For simplicity, E0E and EE0 F2 males have been excluded; see S1 Fig for complete pedigrees.

Fig 3 Fibrotic testis phenotype emerges after multiple generations of estrogenic exposure. (A) fibrotic testis from EEE male showing fusion of the epididymis, vas deferens, and testis. (B) Histological sections of control (top left) and fibrotic testis from EEE male (bottom left; scale bars denote 100 μm); black boxes indicate seminiferous tubules shown in high magnification images in right panels. By comparison with normal testis, fibrotic testis exhibits complete spermatogenic failure, with expansion of interstitial tissue and shrunken seminiferous tubules containing unhealthy Sertoli cells and spermatogonia. (C) Incidence of testicular fibrosis among third-generation males; number above each bar indicates number of males with fibrotic testes out of total scored.

Fig 4 Meiotic recombination levels decrease with successive generations of exposure. (A) Pooled data from exposed families. X-axis represents MLH1 mean of 3 F0 founder males (25-30 pachytene cells/male) and bars show mean ± SEM for E0 and EE F1 sons and E00 and EEE F2 grandsons. Bar color denotes individual exposure (red for exposed, blue for placebo) with increased intensity for successive generations of exposure or placebo. Each group represents data from 25-30 pachytene stage cells/male for 22-23 males. (B-D) Individual data for families 1, 2, and 3 (B, C, and D, respectively); each group represents 4-12 males. Groups were compared by one-way ANOVA; single asterisk denotes significant difference by comparison with founder and double asterisk denotes significance between indicated groups as determined by Tukey-Kramer post-hoc test ($p < 0.05$).

Fig 5 Paternal phenotype affects meiotic recombination levels. Mean MLH1 ± SEM for F1 EE fathers (open circle) and their F2 EEE sons (closed circles). Each point represents the MLH1 mean ± SEM for 25-30 pachytene cells from a single male. Left and center panels show

offspring data from family 2 for two F1 EE fathers with different means: 25.1 ± 0.4 (high paternal MLH1), and 22.6 ± 0.3 (low paternal MLH1). Right panel shows offspring data for EE father with a very low mean, 20.7 ± 0.2, from family 3. Fathers and offspring were compared by one-tailed t-test; for high paternal MLH1, $p < 0.0001$; for low paternal MLH1 $p < 0.01$.

Fig 6 Recombination failure increases with successive generations of exposure. (A) Example of a pachytene spermatocyte immunostained with antibodies to SYCP3 (red) and MLH1 (green), and showing an SC lacking an MLH1 focus (white arrowhead). (B) Frequency of recombination failure in 3 founder males, 28 E0 and 24 EE F1 sons, and 32 E00 and 25 EEE F2 grandsons (25-30 cells analyzed per male).

Supporting Information

S1 Fig Pedigrees of three exposure families. For each family, blue lines of descent indicate placebo and red lines estrogen treatment. Vas deferens phenotype of each male is denoted by square color: normal (white), kinked (light blue), and collapsed (dark blue), with black squares denoting fibrotic testes. Only one EEE and EE0 lineage were obtained in family 1 because the second F1 EE father died in cage.

S2 Fig Frequency of vas deferens abnormalities in individual families. Frequency of normal (light blue), kinked (medium blue), and collapsed (dark blue) phenotypes; each family consists of 9-12 E0 and 8-9 EE F1 sons, and 11-12 E00 and 9-16 EEE F2 grandsons. Comparisons of incidence of abnormal phenotypes: family 1: for E0 and EE, $X^2 = 8.0$ ($p < 0.05$); for E00 and EEE, $X^2 = 11.5$ ($p < 0.01$); family 2: for E00 and EEE, $X^2 = 17.1$ ($p < 0.0001$); for EE and EEE, $X^2 = 7.7$ ($p < 0.01$); family 3: for E00 and EEE, $X^2 = 16.7$ ($p < 0.0001$).

S3 Fig Paternal phenotype affects meiotic recombination levels. Mean MLH1 ± SEM for F0 founders (black) and their F1 EE sons (light red) for families 1, 2, and 3 (left panels). Arrows denote F1 males used to sire F2 offspring. Center and right panels show mean MLH1 ± SEM for F1 EE fathers (light red) and their F2 EEE sons (dark red). Each point represents a single male (25-30 pachytene cells). Fathers and offspring were compared by one-tailed t-test. For family 1: F0 founder v. F1 EE sons ($t = 5.3$, $p < 0.0001$); F1 EE father v. F2 EEE sons right panel ($t = 3.4$, $p < 0.001$). For family 2: F1 EE father v. F2 EEE sons: center panel ($t = 4.6$, $p < 0.0001$); right panel ($t = 3.1$, $p < 0.01$). For family 3: F1 EE father v. F2 EEE sons: center panel ($t = 5.5$, $p < 0.0001$).

S4 Fig Comparison of recombination failure levels in fathers and sons. Recombination failure levels in F1 fathers and their F2 sons for each family. Bar color denotes individual exposure (red for exposed, blue for placebo), increased intensity denotes additional generation of exposure or placebo. Each EE or E0 group represents of 7-11 males (25-30 cells per male); all other groups consist of 7-14 F2 males (25-30 cells per male).

S5 Fig Phenotypic severity is influenced by ancestral and individual exposures. Comparison of 25 F1 EE males and 26 EE0 and 39 E0E F2 males. (A) Frequency of kinked (light blue) and collapsed (dark blue) vas deferens morphology. Incidence of abnormal phenotypes was significantly higher in males with bothe ancestral and individual exposures (E0E): For EE and E0E, $X^2 = 9.7$ ($p < 0.01$); for EE0 and E0E $X^2 = 30.2$ ($p < 0.0001$). Severity of vas defects was also higher in E0E by comparison with EE0 males, $X^2 = 8.3$ ($p < 0.01$) (B) Mean MLH1 ± SEM; 25-30 cells/male. X-axis represents F0 founder mean. Asterisk denotes significant difference as determined using a Tukey-Kramer post-hoc test ($p < 0.05$). (C) Frequency of recombination failure. For EE and E0E, $X^2 = 31.0$ ($p < 0.0001$), and for EE0 and E0E $X^2 = 12.9$ ($p < 0.001$).

S6 Fig Exposure/phenotype by family. Comparison of F1 EE males (n = 8, 9, and 8) and EE0 (n = 4, 12, and 10) and E0E (n = 16, 13, and 10) F2 males from families 1, 2, and 3, respectively. (A) Frequency of kinked (light blue) and collapsed (dark blue) vas deferens morphology. For family 2: EE and E0E, $X^2 = 8.8$ $p < 0.01$; EE0 and E0E, $X^2 = 21.2$, $p < 0.0001$. For family 3: EE and E0E, $X^2 = 4.3$ $p < 0.05$; EE0 and E0E, $X^2 = 6.5$, $p < 0.05$. (B) Mean MLH1 ± SEM; 25-30 cells/male. X-axis represents founder mean. Asterisk denotes significant

863 difference as determined using a Tukey-Kramer post-hoc test ($p < 0.05$). (C) Frequency of
864 recombination failure. For family 1: EE and E0E, $X^2 = 7.2$ ($p < 0.01$); EE0 and E0E, $X^2 = 12.5$ (p
865 < 0.001). For family 2: EE and E0E, $X^2 = 27.1$ ($p < 0.0001$); EE0 and E0E, $X^2 = 11.1$ ($p < 0.001$).
866 For family 3: EE and EE0, $X^2 = 4.9$ ($p < 0.05$).

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from St鋗ane Horel

A TOXIC AFFAIR

How the chemical lobby blocked action on hormone disrupting chemicals

May 2015

STEPHANE HOREL

CEO

EXECUTIVE SUMMARY

Endocrine disruptors are chemicals that are present in everyday products – from plastics and cosmetics to pesticides. Because of their ability to interact with the hormonal (endocrine) systems of living organisms, they are suspected of having severe health and environmental impacts.

EU law demands action be taken on endocrine disruptors, with clear deadlines set. According to these rules, if a chemical is identified as an endocrine disruptor, a ban follows. The current approach is that chemicals are assessed following risk assessment procedures and safe levels of exposure are set accordingly. However, for endocrine disruptors it might be impossible to set such 'safe' levels.

The Directorate-General (DG) for the Environment of the European Commission was put in charge of establishing a set of scientific criteria for 'what is an endocrine disruptor'. The chemical industry lobby was up in arms at the potential banning of some EDCs. The main lobby groups involved were the chemical and pesticide lobbies (CEFIC and ECPA), and the corporations at the forefront were BASF and Bayer. But they found allies in various member states, actors within the European Commission, and in the European Parliament.

The main lobbying tactics used included attempts to undermine and discredit the independent science on EDCs, while promoting industry's own studies as the only 'sound science'; to pressure other Directorates-General in the Commission to go against DG Environment; scaremongering about economic damage industry would suffer; creating delays in the policy process; and using the EU-US trade negotiations (TTIP) as a leverage to prevent any new 'trade barrier'.

By early Spring 2013, since DG Environment did not bend under the pressure, the corporate lobby focused on demanding an impact assessment as a delaying tactic. In a culmination of fierce lobbying pressure, DG Environment's proposal for scientific criteria to identify EDCs was finally rejected by the other DGs in the Commission. Moreover, in July 2013 the Secretary-General, Catherine Day, ordered the impact assessment the industry wanted so much.

This move meant that the Commission failed to meet the December 2013 deadline to come up with the scientific criteria, as demanded by EU law. As the decision process is still ongoing, with the impact assessment on its way, the best-case scenario foresees the final criteria to identify EDCs in 2017.

This report tells the story of how a major EU public health initiative was effectively obstructed by corporate lobby groups in tandem with actors within the European Commission. It shows how industry has successfully used some classic tactics of corporate lobbying. This report shows that some civil servants, even though employed in the services in charge of public health in the European Union, seem to have served corporate interests over public ones.

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TABLE OF CONTENTS

Corporate Europe Observatory (CEO) is a research and campaign group working to expose and challenge the privileged access and influence enjoyed by corporations and their lobby groups in EU policy making. CEO works in close alliance with public interest groups and social movements in and outside Europe to develop alternatives to the dominance of corporate power.

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Stéphane Horel is an independent journalist based in Paris. She investigates corporate influence and conflict of interest on environmental and public health issues. One of her articles on the regulation of endocrine disruptors by the EU was honored by a Laurel of the Columbia Journalism Review. She also directed a documentary on the topic for French TV (Endocrination – What's Up / France 5, 2014).

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Endocrine
Disrupting
Chemicals

Introduction

Endocrine disruptors are chemicals that are present in everyday products – from plastics and cosmetics to pesticides. Because of their ability to interact with the hormonal (endocrine) systems of living organisms, they are suspected of having severe health and environmental impacts. Human exposure to endocrine disrupting chemicals (EDCs) has been linked to diseases such as infertility, cancer and obesity. The medical cost of this serious public health issue has been recently estimated at €157 billion a year in the EU alone.[1] As legislators began to take action, industry has been mobilised for one of EU's biggest lobbying battles.

No less than three pieces of EU legislation demand action be taken on endocrine disruptors, with clear deadlines set: the 2006 regulation on chemicals (REACH), the 2009 pesticide regulation (1107/2009), and the 2012 regulation on biocides (528/2012). Within the European Commission, the Directorate-General (DG) for the Environment was mandated to take the lead. In line with the legislative requirements, DG Environment commissioned a study by independent experts, which was published in 2012. A proposal setting up scientific criteria to define endocrine disruptors, the necessary first step before any legislative action, was set to follow. So far so good? If only.

A TOXIC AFFAIR

HOW THE CHEMICAL LOBBY BLOCKED ACTION ON HORMONE DISRUPTING CHEMICALS

Any potential action taken on endocrine disruptors is a thorn in the side of many industry sectors who see their profits jeopardised. Their efforts to counter any efforts at regulation have mobilised individual companies, lobby federations, and consultancies, both EU- and US-based. These lobbies represent both the chemical industry at large, as well as more specific sectors such as pesticides or plastics producers, that are heavily implicated in the use or manufacture of chemicals suspected to be endocrine disruptors.

In this corporate campaign, multiple lobbying tactics have been used. They include classics such as scaremongering about economic losses, discrediting scientific evidence pointing at the harmful effects of EDCs, and finding reasons to push for delays.

Delaying at all costs any regulations that could possibly deal with EDCs is of crucial importance to the industry because of another (industry-friendly) project the EU has embarked upon: negotiating a free trade deal with the US, the Transatlantic Trade and Investment Partnership (TTIP) – also known as Transatlantic Free Trade Agreement (TAFTA). One of the main goals of this deal is to flatten out the differences between EU and US regulations to facilitate trade. EU action on endocrine disruptors therefore has become a major leverage argument in the negotiations.

This report explores how chemical corporations and their lobby groups – but also actors within the EU institutions – have been working to stop the EU taking action on EDCs, directly endangering public health and the environment.

The decision-maker and the scientist

In 2009, DG Environment was designated *chef de file* in charge of regulating endocrine disrupting chemicals, or EDCs (see box on EDCs next page and box on the regulation below). Its first initiative was to commission a report on the state of science of endocrine disruptors after a call for tender. Prepared by a consortium of experts led by Professor Andreas Kortenkamp of Brunel University, London, the 'State of the art assessment of endocrine disruptors' (from now onwards the Kortenkamp report) was published in January 2012.[2]

The Kortenkamp report is a detailed review of the science on EDCs and several hundred pages long, analysing the most recent body of literature of toxicological and epidemiological studies, and going through the evidence of the effects of EDCs on nature and humans. The authors concluded that any attempt to regulate EDCs would face one major challenge: there is no such thing as a universal, ready-to-use detection kit for EDCs. The reason is that the hormonal system is extremely complex and EDCs can hijack it in many different – and largely unknown – ways.

Indeed, the report identified a wide gap between the increasing knowledge about EDCs, and the way the EU regulates chemicals. They argued that the EU was simply not equipped with the right kind of tests to identify EDCs and pick up their effects. The report therefore recommended some measures to identify and regulate EDCs, in order to address this major threat to public health.

As summarised by Professor Kortenkamp, three elements are needed in order to regulate EDCs:

> "1- Definition (what is it you want to deal with?)
>
> 2- Tests (do you have the tools to identify an EDC?)
>
> 3- Criteria (how to translate test outcomes into regulatory decisions?)".[3]

To these ends, DG Environment started a broad-based policy development initiative. In 2010, it set up an Ad hoc working group involving more than 40 experts from Member States, national regulatory agencies, public research centres, and also representatives of other concerned DGs (Health and Consumers, Research, Enterprise, Employment), of the European Food Safety Authority (EFSA), and other EU bodies. Five 'observer' seats were allocated to industry and NGOs. In addition, DG Environment created an 'Expert Advisory Group' the following year, to provide technical advice on the development of the criteria.[4]

Within both working groups, a consensus quickly emerged accepting the World Health Organisation / International Programme on Chemical Safety (WHO/IPCS) definition of EDCs: "An endocrine disruptor is an exogenous substance or mixture that alters function(s) of the endocrine system and consequently causes adverse health effects in an intact organism, or its progeny, or (sub)populations".[5] With this definition in place, attention shifted to the identification criteria of endocrine disruptors, which turned into a battleground.

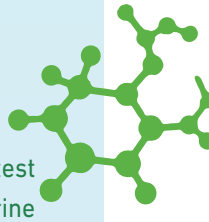
Box I

The legal provisions

The 2009 pesticide regulation established "hazard-based cut-off criteria" for EDCs. As the law considers EDCs hazardous, pesticides with endocrine disrupting properties will no longer be authorised on the EU market. This 'hazard-based approach' replaces traditional risk assessment that – as described in Box 2 – aims to define a 'safe' level of exposure. The EU Commission had to develop a scientific definition and criteria to identify EDCs before 14 December 2013.[6]

As also required in a provision of the 2006 REACH chemical regulation, the EU Commission had to decide on whether thresholds can be set for EDCs or not. If there are no safe thresholds and EDCs are dangerous whatever the concentrations, then these chemicals would eventually have to be substituted or simply banned. If alternatively, thresholds do exist and EDCs are considered safe under a certain concentration, they would be left on the market. This decision was to be taken before 1st June 2013.[7]

The chemical industry lobby groups strongly oppose the hazard-based approach of the pesticide regulation. They argue that EDCs can be regulated like any other chemical through the current system of risk assessment.[8]



Box 2

Endo-what?

Endocrine disrupting chemicals (EDCs) can interfere with the hormonal systems of mammals, fish, frogs, and other types of living organisms. Their toxicity only started to be fully acknowledged by scientists in the early 1990s. EDCs have the ability to mimic, block, or alter the levels of hormones such as oestrogens, testosterone, or thyroid hormones whose actions affect many functions of the body. Exposure to these chemicals in the early developmental stages of an organism can cause irreversible effects that will only become evident later in life.[9] There is a high probability that EDCs play a role in the genesis of many 'modern' diseases such as prostate, breast, and testicular cancers, infertility, genital abnormalities, brain development, diabetes, and obesity. Nearly 1000 substances have been identified so far as potential endocrine disruptors,[10] but it could be double that amount.[11] EDCs are found in widely used products such as pesticides, plastics, cosmetics, carpets, computers, and construction materials. They end up in food, air, dust, rivers, oceans, wild animals and... our bodies. One example of an EDC, bisphenol A (BPA), has already been banned from baby bottles in the EU over health concerns.

In 2013, a major report underlined the urgency of taking action on EDCs. The 'State of the Science on Endocrine Disrupting Chemicals' was published jointly by the World Health Organisation (WHO) and the United Nations Environmental Programme (UNEP), and highlighted that the vast majority of chemicals already on the market have never been tested for potential endocrine disrupting effects, while international test methods capture only some of the known endocrine disrupting effects. EDCs represent a "global threat that needs to be resolved", the WHO/UNEP report concluded.[12]

The report also stated that the exposure of both humans and wildlife to such chemicals comes from an increasing number of sources, and that the risk from mixtures of these substances – the so-called 'cocktail effect' – is severely underestimated. The report underlined that these effects may occur below established safety levels for individual chemicals.

When chemicals are regulated (but many of them are not), they are assessed on the assumption that there is a 'safe level of use'. Thresholds are set below the "no observed adverse effect level" (NOAEL). It is however accepted that some chemicals do not have a 'safe level' or threshold that people can be exposed to. It is the case with some carcinogenic, mutagenic and reprotoxic chemicals (CMR) and also with persistent, bioaccumulative and toxic substances (PBTs). The question of a 'safe' threshold is at the core of the debate on EDCs. Yet according to one of the most detailed reviews of the science on EDCs to date, an authoritative report by a team led by Professor Kortenkamp for the European Commission (see next section), the current tools we have are not adequate to detect thresholds for these chemicals.[13] This would imply that EDCs should be regulated as "non-threshold" chemicals.

The Kortenkamp report recommended a list of criteria that would complement each other, such as adversity, mode of action, potency, lead toxicity, specificity, severity, irreversibility, and relevance. No criterion, the report stated, should be used in isolation as a cut-off filter.[14]

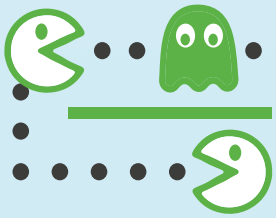
But in May 2011, the British and German authorities published a joint position on the EDC criteria.[15] Making no secret of their concern for the "great commercial impact" of the EDC regulation, the two Member States defended a cut-off criterion that would filter out only the most "potent" EDCs. The idea behind this proposal, explained Professor Kortenkamp, "would be to use the criterion of potency as a tool to cream off from the top the 'worst offenders' and leave the rest of EDCs totally unregulated."[16] The Kortenkamp report clearly stated that such potency values were "largely arbitrary and not scientifically justifiable". Yet this was not a problem for the two Member States.

The inclusion of potency as a cut-off criterion could indeed spare a significant number of pesticide products from a ban. So it became a key lobbying demand of the chemical and pesticide industries. This idea was subsequently developed in a scientific article published in October 2012 in an industry-owned journal.[17] The article was sponsored by ECETOC (European Centre for Ecotoxicology and Toxicology of Chemicals), an industry-science organisation, whose members include BASF, Bayer, Dow, and Syngenta.[18]

Box 3

Corporate lobbyists tricks and ploys

The corporate lobby against the EDC regulation employs numerous tools from the lobbyists' toolbox. These include:

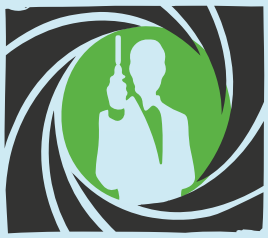


Isolating the 'good guy'

Mobilise actors in the European Commission, such as DG Trade, DG Enterprise and the Secretariat General, against DG Environment.

Scaremongering with economic losses

Lobby groups produce dramatic figures to tell the EU institutions how bad the economic impact on their sector will be.



Hired-gun lobbyists

Specialised lobby consultancy firms (also called public relations / public affairs companies) and law firms are hired by corporations or sectoral lobby associations to develop strategies, broker meetings with officials or decision makers, etc.

Undermine the science

Orchestrate and fund 'critiques' of the Kortenkamp report.

Delay and derail

By asking for an impact assessment, industry aims to buy time hoping to get EDC regulation off the table for good later.

Using 'free trade' agreement to undermine EU regulation

TTIP negotiations are aiming to align food and environmental safety rules with the US, which would in many instances lead to a downgrading of EU rules.

Mobilising 'other voices' to repeat the message

Fund or otherwise support scientists, farmers organisations or other to join the chorus in attacking EDC criteria.

Box 4

Who are the lobbyists?

Brussels nowadays is the second capital of corporate lobbying in the world – after Washington DC. An estimated 20-30.000 lobbyists populate the EU quarter, the large majority of whom represents corporations.[19] All big corporations have their own lobby offices and in-house lobbyists.

But orchestrated campaigns such as the one about the endocrine disruptor criteria often happen through industry associations representing different sectors: in this case CEFIC (European Chemical Industry Council) and two of its spin-offs ECPA (European Crop Protection Association), and PlasticsEurope; and also Cosmetics Europe. ECPA's president is Martin Dawkins of Bayer. CEFIC's leadership team is dominated by (current and former) BASF people. PlasticEurope's president Patrick Thomas is the CEO of Bayer MaterialScience AG – one of the main world producers of bisphenol A.

The interests of US industry are well represented in Brussels. Most pesticide corporations are members of Croplife America, ECPA's sister organisation. Their interests are also defended by the American Chamber of Commerce (AmCham EU) that closely works with Brussels-based PR firm EPPA. Specialised 'hired-gun' lobby consultancy firms (also called 'public relations' (PR) or 'public affairs' (PA) companies) are contracted for particular jobs to support these corporations' interests.

Then there are those industry lobby platforms that aim to get business interests promoted in scientific debates and fora, such as ECETOC (European Centre for Ecotoxicology and Toxicology of Chemicals). ECETOC is described on its website as an "industry-funded expert not-for-profit think tank" whose purpose is "to enhance the quality of chemicals risk assessment".[20] Bayer, BASF, Dow, DuPont and Syngenta are among the many corporations member of ECETOC.[21]

Box 5

Big budgets

Access to reliable, high-quality information on lobbying in the EU is sparse. The European Transparency Register is voluntary, and information is not checked. According to the register, the CEFIC alone reported a total budget of 40 million euros in 2012, of which they reported to have spent 'only' 6 million on lobbying.[22] But defending their corporate members' interests in Brussels is their sole *raison d'être*. ECPA claims to spend merely between €50,000-100,000 per year on lobbying.[23] They only count the gross salary costs of the number of hours their lobbyists spend being present at the Commission, the Parliament or an agency like EFSA, plus some overhead costs. These self-reported figures do not represent the true lobbying costs.

Box 6

Revolving doors

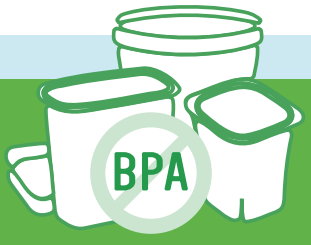
Lobby groups often employ the classic tactic of the 'revolving door': in other words, to hire people who come straight from a job in government. Many lobbyists are former Commission officials or Members of the European Parliament, or Parliament or Council staff. They are therefore in a good position to then lobby their former colleagues, and they know how the system works from the inside. The revolving doors can also spin in the other direction, that is, when someone from within the industry moves to a key position in a public authority.

The pesticide lobby has many examples. Looking at ECPA's current staff: Stuart Rutherford used to work for DG Environment and Agatha Pietrasiuk at DG SANCO on pesticides, while Jess O'Flynn, Michal Kicinsky and Anna Gatt Seretny are all former MEP assistants.[24] O'Flynn worked for British conservative MEP Julie Girling until the last European elections in 2014.[25] CEFIC lobbyist Lena Perenius previously worked in DG Enterprise on the chemical package REACH.[26] As for Ralf Burgstahler, he started at BASF, moved on to work in the European Commission on REACH (which BASF aimed to undermine), then took a position in a German ministry, and is now back at BASF as a lobbyist where he works on plasticisers (such as phthalates, known to be endocrine disruptors).[27] [28]

In the US, the use of the revolving door is even more common. According to the Centre for Responsive Politics, more than half of CropLife America's lobbyists in the period 2013-2014 previously held government jobs.[29]

Box 7

The controversial case of bisphenol A



The chemical bisphenol A (BPA) is the most well-known example of an endocrine disrupting chemical.[30] It is primarily used to make shatter-proof polycarbonate plastics, and has been found to leach from these materials. It has been banned from baby bottles in the EU since 2011.[31] But BPA is still widely used in consumer products such as the inside of food and beverage cans, dental fillings and thermal paper for tickets. The French food safety authority ANSES concluded in 2011 that health effects from BPA had been proven in animals and suspected in humans, even at lower levels of exposure than the 'safe' dose allowed by EFSA.[32] Yet EFSA comes to different conclusions, including in its last review and opinion on BPA from January 2015, which provoked renewed criticism. In a response, French Environment Minister Ségolène Royal openly wondered what weight the industry had had over EFSA in this case.[33]

Attacks on the Kortenkamp report

The main conclusions of the Kortenkamp report annoyed industry. Attacks soon followed. The first was a "critique" published in May 2012 in a peer-reviewed scientific journal.[34] It was sponsored by the American Chemistry Council, the lobbying organisation for the US chemical industry. All five authors worked as consultants for industry, two of them being employed by Gradient Corp, a product-defence company which performed studies on a known endocrine disrupting chemical (bisphenol A) on behalf of industry,[35] and has had Bayer amongst its clients.[36] Another industry critique was commissioned by ECETOC to Exponent, also a US-based product-defence company.[37,38] The third attack came from... the UK Government. In July 2012, the UK Department for Environment, Food and Rural Affairs (Defra) released an unsigned, 3-page long "comment" from its Hazardous Substances Advisory Committee (HSAC) criticising the Kortenkamp report's methodology.[39]

As early as mid-2012, DG Environment's reluctance to take the wishes of industry on board had become quite evident. DG Environment was however facing mounting pressure. From the British and German governments, from industry, but also from inside the Commission itself.

The EFSA plot

On 1st October 2012, in a surprise move that undermined DG Environment's position, EFSA announced that it had been tasked by the European Commission with forming a scientific opinion on "the human health and environmental risks associated with the possible presence of endocrine disruptors in the food chain".[40] This meant an opportunity to give their view on the issue of scientific criteria for endocrine disruptors as a whole.

The official mandate to EFSA was signed on 1st August 2012 by the Director General of DG SANCO Paola Testori Coggi.[41] DG Environment was not copied in on the official mandate and was only informed a few days later.[42] With this hostile gesture, DG SANCO sidelined DG Environment in an attempt to take some control over the development of the EDC criteria. Professor Kortenkamp confided that some of his colleagues expected EFSA to "come out in favour of potency based cut-off values, as proposed by industry and some Member States".[43]

Was EFSA even the most adequate body to give a thorough scientific judgement on EDCs? EFSA's previous work on bisphenol A had been controversial and criticised for instance by the French Agency for Food, Environmental and Occupational Health & Safety (ANSES).[44]

EFSA went to work and formed a working group on EDCs. Soon after, a media report showed that 8 out of 18 members of the EFSA working group on endocrine disruptors had conflicts of interest. Three of them had ties with industry lobby group the International Life Sciences Institute (ILSI), one with CEFIC, another with Syngenta.[45]

Moreover, the EFSA group included three experts employed by the British and German administrations – which had already taken (pro-industry) sides in the potency criteria debate. Finally, only 4 out of 18 experts had done actual scientific research on endocrine disruptors. None was a specialist in human endocrinology.

So what did this EFSA working group come up with?

Box 8 What is a product-defence company?

Product defence companies are in the (big) business of shaping and skewing science to the liking of their clients. These companies employ scientists to perform studies, to produce data that suit the client's interest, or to criticise studies that don't. David Michaels, author of the reference book "Doubt is their Product"[46], said about product defense firms: "I have yet to see a study published by a product defense firm that conflicts with the needs of the study's sponsors. The intent is to cast doubt on real science."[47]

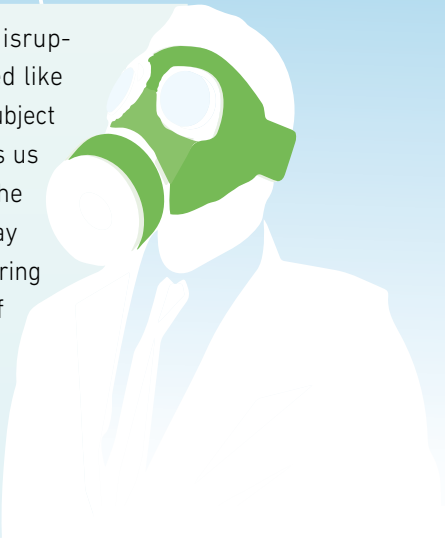
Two emails have surfaced, obtained through an access to documents request, that strongly suggest that at least one member of the EFSA working group had doubts about EFSA's opinion on EDCs shortly before it was published. The email relates to the fact that on 19 February 2013, an authoritative report on EDCs was published by the World Health Organisation and the United Nations Environment Programme (UNEP).

In an email sent on the following day, this working group member expressed grave concern to his (or her) colleagues and to EFSA staff supervising their work, about the quality of the group's own work:[48]

> "Dear colleagues,
>
> Life is complicated...
>
> It is almost embarrassing to compare our current draft report with the WHO-UNEP report. The issues the WHO-UNEP report highlight and takes out as being specific for [endocrine disruptors], we in our report are trying to down-play or even avoid, when WHO-UNEP comes to the conclusion that traditional risk assessment of chemicals is not fit for purpose to assess [endocrine disruptors] (p 17), we are exactly coming to the opposite conclusion.... [T]hey discuss elegantly why "thresholds" could not be applied to [endocrine disruptors]. We stay at the best "luke-warm" to these issues.... I am happy I don't need to be at the press conference and stakeholder meeting (as planned the 20 March) and present and defend the current EFSA [Scientific Committee] report knowing that the audience have read the WHO-UNEP report. A straightforward killer situation!...
>
> I cannot see any other way out of this than we have to re-do our report or at least significantly modify it....
>
> We could wonderfully have used the WHO-UNEP report as a next step way forward in identifying for [endocrine disruptors], with all the precautions and restrictions it takes. Unfortunately we did not do this and now we are in a mess!"

Here is what Bernard Bottex, the EFSA staff member supervising the EDC working group, replied:

> "[We need to] reconsider our conclusions: options 2 and 3 of the current conclusions where we explain that [endocrine disruptors] should be considered like most other chemicals, ie subject to a risk assessment, puts us in isolation compared to the rest of the world, and may be hard to defend considering the uncertainties, lack of data and methods identified. Any suggestion for rewording based on these new parameters will be welcomed."[49]



When asked for a comment, EFSA replied that these emails took place within a wider scientific discussion and therefore "should not be seen in isolation". Other experts expressed contradicting views, EFSA said. EFSA added that the scope of the WHO/UNEP report "allowed for a deeper discussion" on issues like the low-dose effects, and that EFSA has now commissioned a new study into those effects.[50]

EFSA's opinion was finally published on 20 March 2013.[51] Despite the misgivings the above email expresses, the concluding sentence of EFSA's opinion that Bottex proposed to modify finally remained unchanged. EDCs "can therefore be treated like most other substances of concern for human health and the environment, i.e. be subject to risk assessment and not only to hazard assessment", said the conclusion. As explained earlier in this report, a hazard assessment of a chemical identifies its potential hazards such as endocrine disrupting properties. Risk assessment can then follow, establishing a 'safe level of use', but it is questioned whether this is at all possible for endocrine disruptors.

Yet EFSA had managed to somehow dodge the issue: the sentence does not *request* that EDCs should be subjected to risk assessment. And there's a good reason for this. The EU pesticide regulation prescribes a hazard-based approach for EDCs – so not a risk assessment approach. An EU agency, EFSA would probably not want to be seen to contradict the European law.

Although EFSA's opinion contained such inconsistencies and other problematic aspects, it nonetheless did not propose or advocate the industry's desired potency cut-off criterion. If it had been DG SANCO's intention to force DG Environment to include the latter by having EFSA legitimising it, then this attempt had ultimately failed.

Meanwhile in the Parliament

Endocrine disruptors had become a topic of debate in the European Parliament too. In April 2012, an own-initiative report had been started, led by Swedish Socialist MEP Åsa Westlund. This report supported the precautionary approach already taken by DG Environment. Westlund confirms that, especially in the beginning, she received numerous phone calls and emails from chemical industry lobbyists: "Industry tried to confuse the debate and shift the attention to phasing out only the most dangerous chemicals. But first you have to know what the most dangerous endocrine disruptors are!" she said.[52]

Westlund's work was directly challenged by British Conservative MEP Julie Girling (European Conservatives and Reformists Group). Girling is the agriculture spokesperson for the Conservative Party in the UK. She makes no secret of her views favourable to industry interests when it comes to issues like pesticides and GMOs.[53,54]

In September 2012, Girling set up an 'Informal Working Group on risk-based policy making'. On her website, she is said to be concerned that too many decisions are based on "ultra-cautious responses to perceived hazards rather than a rational and science-led examination and measurement of real risk".[55]

On behalf of this informal working group (which is unknown to have other members), she organised a closed event entitled "Risk versus Hazard – with reference to the Westlund report on Endocrine disruptors" scheduled for 22 January 2013. Girling wrote to the Chief Scientific Adviser of the President of the EU Commission Anne Glover to invite her as a guest speaker. In her letter, Girling called Westlund's report a "good example of how risk is being neglected when it comes to making policy decisions in the area of chemicals legislation". She promoted the event as a "chance to meet some of those supportive of risk-based policy-making".[56] Again, "risk-based policy making" here refers to the approach that a 'safe level of use' can be established for any chemical.

Glover accepted the invitation to be a guest speaker, along with others such as EFSA Director of Science Strategy and Coordination Hubert Deluyker, and Rémi Bars, a toxicologist for Bayer and also chair of ECETOC. The "confirmed guest list" forwarded to Glover gives a clear picture of who was seen by Girling as "supportive of risk-based policy-making". The list included representatives of numerous chemical lobby outfits in Brussels: CEFIC, ECPA, PlasticsEurope, Toy Industries of Europe, someone representing Bayer and ECETOC, BASF, ExxonMobil, the American Chamber of Commerce (AmCham EU), and PR firm Burson Marsteller; but not a single environmental or public health NGO. And what's more, not even Westlund herself, who – despite the fact that her name was in the title of the event – was invited.[57]

In the run up to a vote in the Environment Committee on Westlund's report, MEPs Julie Girling and Miroslav Ouzky (both from the European Conservatives and Reformists Group - ECR) jointly tabled 22 amendments to the Westlund report. Their changes for instance aimed to get the precautionary principle out of the text, and replace it with promotion for classic risk assessment.[58] MEPs who tabled similar amendments in order to weaken Westlund's text included Oreste Rossi (from the right-wing and Eurosceptic Europe of Freedom and Democracy Group - EFD), Pilar Ayuso (from the also Conservative Group of the European People's Party - EPP), Cristina Gutierrez-Cortines (EPP) and Andres Perello Rodriguez (from the Socialists and Democrats Group - S&D).

Nonetheless, Westlund managed to secure a large majority of support for her resolution in the Parliament and it was adopted on 14 March 2013, just a few days before EFSA's opinion was published. It unambiguously stated that the precautionary principle "require[d] the Commission and the legislators to take adequate measures to reduce short- and long-term exposure of humans to endocrine disruptors". In opposition to what industry, the UK and Germany had been saying, the resolution also stressed that "no single criterion should be seen as cut-off or decisive for the identification of an endocrine disruptor". In short, it meant that the potency criterion should be discarded.

Lobbying offensive, first round: the impact assessment

Early spring 2013 was a turning point. The WHO/UNEP report on endocrine disruptors had been published in February, stating that EDCs were a "global threat that needs to be resolved". There was the Parliament report led by Westlund supporting the work accomplished by DG Environment. Then, relying on the Kortenkamp report and on EFSA's opinion, DG Environment's expert group published their own final report.[59] DG Environment's services began finalising a proposal for the identification criteria of EDCs.

The chemical industry realised they did not hold a winning hand. Their strategy to have only a ban on the most potent endocrine disruptors seemed doomed to fail. They now became seriously alarmed and were looking for a way to throw a spanner in the works: in this case, to create a delay. An ideal tool for this is to request an impact assessment. This administrative procedure, which takes minimum 12 months, aims at evaluating the positive and negative impacts of a Commission policy proposal. History has shown that the outcome is more susceptible to favour economic interests than anything else (see Box Assessing What impacts, exactly?). This is what the toxic lobby decided to go for.

In Spring 2013, the industry lobbying campaign for an impact assessment took off in full force (See Annex I for examples). Special targets in the Commission were the Directorates-General SANCO, Enterprise and Trade, and also the Secretary General. The aim: to secure their support and to isolate DG Environment's pocket of resistance. Of course, they would be quick to present their own, alarmist figures on what the EDC criteria would mean for their industry. It is indeed a known classic strategy for industry to 'cry wolf' and overestimate the costs of new forthcoming environmental or public health legislations, never taking into account the (financial and non-financial) benefits that it produces.[60]

In March 2013, the pesticide industry lobbying organisation ECPA produced a document assessing the economic impact of the draft criteria for endocrine disruptors.[61] It relied mostly on an impact assessment performed in 2009 by the UK government and contained some alarmist claims. The criteria would "severely reduce the availability of crop protection products in Europe", it said. The market value of products that could be affected by the EDC criteria was "calculated at between €3-4 billion". The yield loss of key crops such as wheat, potatoes, oilseed rape and vines would be "between 10-20% in an average year – with losses of up to 50% being possible in years of high disease pressure". In addition the criteria would of course severely hamper "global commerce". This key lobbying document was subsequently spread widely among Commission officials.

Early June 2013, DG Environment's proposal went into the final phase before being published. Up until the very last moment, industry tried every possible opening to apply pressure. Illustrative of this is an almost desperate attempt at the end of May 2013, by AmchamEU and EPPA (the lobby firm they hired) to get to see the Chief Scientific Adviser, Anne Glover, even though she had no immediate say in the matter. EPPA's Miglena Mihova, chair of AmChamEU's Environment Committee asked her for "even just 15 minutes", "to share some of industry's concerns".[62]

Box 9

Assessing what impacts, exactly?

Even if impact assessments are supposed to evaluate "the potential economic, social and environmental consequences" of a Commission initiative,[63] the outcomes are more likely to be in favour of the economic aspects rather than the public health or environment aspects, for the simple reason that the latter are much more difficult to evaluate. "We know from the history of previous efforts to do cost-benefit analyses, impact assessments, that they're deeply flawed generally speaking because it is much easier to put numbers on costs of regulation than it is to put numbers on what are the benefits to society over the next four or five decades of not having reproductive problems," said David Gee, former Senior Advisor on science, policy and emerging issues at the European Environment Agency (EEA).[64] Conveniently for industry, an impact assessment takes on average 12 to 15 months and so can also work as a handy delaying tactic.

On 7 June at 9.30am sharp, all the concerned Commission Directorates-General were invited by DG Environment to comment on their draft criteria proposal.[65] The meeting, called 'interservice meeting', was a make or break moment. But by lunchtime, the draft was refused as it stood, and the rupture was effective.

From what happened thereafter, one can safely assume that DG Environment's proposal had already been leaked to the outside world previous to that meeting. That very same day of 7 June, at 2.04pm precisely, chemical giant Bayer sent a well-targeted email to the highest level in the Commission, the Secretariat General. The recipients were Marianne Klingbeil and Stefan Moser. Marianne Klingbeil is Deputy Secretary General, and responsible for the EU impact assessments. Bayer, writing in German to a fellow countrywoman, put forward both the UK impact assessment and a similar report by Teagasc, the Irish Agriculture and Food Development Authority, about the impact of "an inappropriate endocrine disruption definition upon wheat disease control programmes and production in Ireland". "Despite the massive impacts on the combined industry and agriculture sector", Bayer complained, "the Commission has so far refused to undertake an impact assessment. We therefore ask you to stand up for the implementation of an impact assessment".[66]

In the weeks to come, the lobbying supporting the impact assessment only intensified further.

Examples of lobbying emails sent by industry to various targets in the European Commission.

Emails are available from Corporate Europe Observatory and Stéphane Horel.

Would the Commission bow to the demands for an impact assessment? First, we need to take a look at the chemical industry's parallel lobbying route against any EDC regulation: the EU-US trade negotiations.

Lobbying offensive, 2nd round: the TTIP

Industry was in the meantime presented with a second, unique opportunity for a fruitful lobby against EDC regulation: the upcoming negotiation of a free trade deal between the EU and the US, known by its acronyms TTIP (Transatlantic Trade and Investment Partnership) or TAFTA (Trans-Atlantic Free Trade Agreement). One of the main goals of the TTIP is precisely to iron away the differences between EU and US regulations in order to facilitate trade flows. A regulation of EDCs would present a major new difference in rules between the two blocks. These negotiations were therefore latched on by industry as the perfect opportunity to get rid of the EDC issue altogether.

On the US side, the American Chemistry Council (ACC) and CropLife America (CLA) wrote to the US Office of Chemical Safety and Pollution Prevention (OCSPP) at the end of 2012, saying they have "serious concerns" that the EU EDC criteria will "would trigger negative and far reaching impacts on global commerce."[67] They warned that the adoption of an approach in the EU that differs so substantially from the US approach would "likely put in place precisely the kind of regulatory barriers that a potential US-EU Free Trade Agreement would be designed to address".

In March 2013, an unnamed consultant organised several meetings in Brussels for a delegation of the US pesticide lobby group CropLife America. According to an email he sent to Jean Ferriere of the Secretariat General of the European Commission, their main concern was the forthcoming regulation of EDCs which did not "appear to be consistent with the objectives of the US-EU negotiations for a TTIP".[68] The CropLife America delegation was invited to join a meeting already planned with ECPA on 20 March at the Secretary General.

Their demands were clearly formulated in a Croplife America position paper:[69]

> "• The hazard based cut-off criteria in EU Regulation 1107/2009 should not impact U.S.-EU trade;
>
> • The EU's use of suspension or bans of products to control product uses while avoiding risk assessments should not impact U.S.-EU trade;
>
> • The U.S. Government should defend itself using authority of the [Sanitary and Phytosanitary Measures] Agreement under [Word Trade Organisation], if the EU pursues its proposed new regulatory regime for endocrine disruptors without an approach based on risk assessment."

Another key industry demand for TTIP is 'regulatory cooperation' (see Box 10). In June 2013, a delegation of the American Chamber of Commerce (AmCham EU) met with officials from DG Enterprise and DG Trade to discuss what regulatory cooperation could look like.[70] DG Trade imagined a mechanism that would make it obligatory to provide a justification, in the event that either the EU or US wished to create a new regulation that the other party disagreed with.

Commission-industry symbiosis – push for 'regulatory cooperation'

Evidence shows that if industry does not do a good enough job at lobbying by itself, DG Trade will help them with a gentle nudge. In autumn 2012, DG Trade chased pesticide lobby group ECPA to participate in the then-ongoing public consultation on TTIP. As the European pesticide industry is "one of the key sectors we would be looking at in terms of improving the framework for business," DG Trade emailed ECPA, "your contribution, ideally sponsored by your US partner, would be most welcome".[71] ECPA responded a few weeks later, together with its US sister organisation CropLife America, demanding for instance the harmonisation for pesticide residues in food, and pushing for 'regulatory cooperation'.[72]

Box 10

Regulatory cooperation is a tool that would prevent differences in standards in the future. This could represent a major new threat to any legislation aiming at protecting health and the environment.

Shortly after, the chemical lobby (CEFIC and American Chemistry Council) proposed a very similar idea of a "Joint Scientific Advisory Council" to deal with "emerging" issues such as EDCs".[73]

On 17 June 2013, José Manuel Barroso and Barack Obama announced the official launch of the negotiations over the EU-US trade agreement[74].

Since summer 2013, lobbying has gone on unabated to use TTIP against any regulatory action on EDCs. But DG Trade has an increasingly hard time in selling TTIP to the European public. DG Trade official Jean-Luc Demarty pleaded to CEFIC that more vocal industry support was needed "in passing the message that TTIP is not about dismantling existing EU chemicals legislation."[75] The US Government has been more straightforward about its role as industry agent; its trade department explicitly singled out the regulation of endocrine disruptors as a 'trade barrier' that should be removed through TTIP.[76] Industry also often requests that EU regulations should be based on "sound science". This flagged expression was coined by the tobacco industry (see Box 11).

Concerned scientists or industry front group?

While the industry lobbying offensive peaked this month of June 2013, another voice joined their chorus.

On 17 June 2013, a group of 56 scientists led by German toxicologist Wolfgang Dekant sent a letter to Barroso's Chief Scientific Adviser Anne Glover, attacking the work done by DG Environment on EDCs.[77] "The currently drafted framework is based on virtually complete ignorance of all well-established and taught principles of pharmacology and toxicology", they asserted, yet without referring to any precise document. The letter remained unknown to the public until 5 July 2013, when it was published online in a toxicology journal together with an editorial nailing their point. Entitled 'Scientifically Unfounded Precaution Drives European Commission's Recommendations on EDC Regulation, While Defying Common Sense, Well-Established Science and Risk Assessment Principles', the editorial was signed by a further 18 editors and

The rhetoric of 'sound science'

From tobacco in the 1950s to climate change today, there is now a long history of industry attempts to "manufacture doubt"[78] over scientific evidence that shows harmful effects of their products. One way to do this is for instance to fund studies that point at other possible causes for these harmful effects. Industry would then claim their studies to be 'sound science', while the inconvenient studies are labelled 'junk science' (other variations used are 'not science-based' or 'not evidence-based').

With the arrival of TTIP, industry is recycling this Orwellian notion of 'sound science' to stage an ongoing attack on the EU food safety system, including the precautionary principle. For instance, ECPA and CropLife demand "the inclusion of science-based risk assessment as the unified basis for pesticide regulation",[79] implying that the EU pesticide regulation is not science-based.

One example of a political figure buying into industry's framing is the British Conservative MEP Julie Girling. In an opinion piece in the *Wall Street Journal* titled "The Junk Science Threat to Free Trade", she wrote that the biggest threats to TTIP's success were the endocrine disruptors issue and the use of the precautionary principle in the EU. She described the evidence of harm of EDCs (and other classes of chemicals) for human health as "hypothetical at best, possibly illusory, and certainly never scientifically established." Endocrine disruptors are now "stigmatized by anti-chemical activists", she continued. She ended by saying that Europe needs to move to "a system that assesses real, known impacts based on sound science".[80]

However, there is overwhelming evidence showing that government action against harmful substances – from asbestos to lead, and from tobacco to some pesticides, has been delayed for years, sometimes entire decades because of industry lobbying undermining the science.

Box II

associate editors of scientific journals led by Daniel Dietrich, a toxicologist at the University of Konstanz.[81] The editorial would thereafter be published in no less than 14 journals in the course of the following months. This *modus operandi* was unseen before in the history of scientific literature.

Considering the timing of this very unusual letter and editorial, coming in the heat of the fight of industry against action on EDCs, many eyebrows were raised. Its content did not go unanswered either. At the end of August 2013, the first rebuttal was published in the journal *Environmental Health* by 41 leading experts in endocrine disruption, four of whom had participated in the landmark WHO/UNEP 2013 report, and two in the Kortenkamp report. "We are concerned that the Dietrich editorial appears to be intended as an intervention designed to impact imminent decisions by the European Commission concerning endocrine disrupting chemicals", they wrote.[82] The second rebuttal was published a couple of weeks later in the journal of the Endocrine Society, this time signed by 104 scientists and editors of journals. The editorial, they concluded, "does the European Commission, science, including the field of toxicology, and most importantly, public health, a profound disservice".[83]

Shortly after, an investigation by *Environmental Health News* reported that out of the 18 editors who signed the Dietrich editorial, 17 had ties with the chemical, pesticide, cosmetics, pharmaceutical, biotechnology, or even tobacco industries. As far as the chemical industry is concerned, links could be identified with the American Chemistry Council, CEFIC, ECETOC and ILSI.[84] Furthermore, among the 56 original signatories of the letter to Anne Glover, at least 33 also had industry ties.[85] Some had received research funds from industry associations, while some had served as industry consultants or advisors.

Remarkably, the letter to Chief Scientific Adviser Anne Glover was signed by three scientists – namely Diane Benford, Gisela Degen, and Josef Schlatter – who also happened to be members of the 2012-2013 EFSA working group on EDCs. Benford, Degen and Schlatter were all three among those found to have conflicts of interest with the commercial sector.[86]

The letter undermining DG Environment's work seemed to hit home. Only three days after she received the letter criticising the EDC criteria, Anne Glover sent a note to Karl Falkenberg, the Director General of DG Environment. She had received a letter from "a large number of very eminent experts in the field of toxicology", she wrote, presenting them as authoritative on the EDC issue. Then she demanded explanations on the process: how the "evidence was reviewed"? Why was EFSA's opinion "ignored"? Was it true that the EDC regulation "would be based solely on the base of *in vitro* tests"? The tone was not very amiable.[87]

But most importantly, Anne Glover copied her note to the cabinet of Barroso and to the Secretary General Catherine Day. Passing on the impression that there were legitimate reasons to question the scientific work performed by DG Environment, she rang the alarm bell at the upper floors of the Commission at a crucial moment.

The decisive blow

On 2 July 2013, the decision process on the EDC criteria was finally derailed. The Commission Secretary General Catherine Day wrote a note to both Karl Falkenberg and Paola Testori Coggi, the Directors-General of DG Environment and SANCO respectively, ordering them to work together on the EDC criteria, demanding that the proposal "should be supported by an impact assessment including a public consultation on the various options for the criteria and their impact".[88]

The issue, further argued Catherine Day, is "sensitive because of the diverging views held by the stakeholder community and the potential impacts on parts of the chemical industry and international trade". As various services of the Commission had been methodically fed with industry-commissioned and UK impact assessments, and warnings over the TTIP, the concern over "the potential impacts on parts of the chemical industry and international trade" can be easily explained. But what about the "diverging views held by the stakeholder community"? Which diverging view could that be except for the motley crowd of scientists, whose critique had been given weight and credit just a couple of weeks earlier by Anne Glover's intervention?

As acknowledged later by the Commission, industry lobbying and the letter by the scientists had indeed been the decisive factors for this outcome.[89]

With this decision to launch an impact assessment, the Secretary General had single-handedly thrown a monkey wrench in DG Environment's work on EDCs. Not to mention that the process would *de facto* be delayed for an undefined period of time regardless of the legal deadline – December 2013 – set by the Parliament. Industry had managed to buy the time they needed to try to weaken the criteria, and to benefit from the deregulatory, 'free trade' dynamic offered by the EU-US trade talks. The businesses with most to lose from regulation of EDCs could celebrate.

In early September 2013, the decision to make an impact assessment on the EDC criteria was finally made public. A group of eight MEPs following the issue closely responded with a letter to the then-President of the European Commission Barroso: "This decision is surprising, as one would expect scientific criteria to be based on objective scientific studies and not on an impact assessment, which is rather a tool to inform political decisions." In other words: if the aim is to develop a scientific definition of what an EDC is, then the potential economic (or other) impacts are completely irrelevant. The MEPs commented: "Doing an impact assessment from the outset seems to confuse science with policy-making and hazard with risk."[90]

The surprise consensus package

On 24 October 2013 nonetheless, Chief Scientific Adviser Anne Glover convened a meeting in her office with representatives of the two scientific 'camps'. The camp who had criticised DG Environment's work with the letter to Glover, featured Alan Boobis, Wolfgang Dekant and Helmut Greim. The 'EDC scientists camp' – Anna Maria Andersson, Ulla Haas, and Andreas Kortenkamp. Nobody expected the confrontation would have such a surprising outcome: the critics' group radically changed their position. They agreed to sign a consensus statement, which contradicted their initial declarations, notably on the issue of whether there were safe thresholds for EDCs. "It is possible that thresholds do not exist", and "it is not possible to define thresholds only by experiments in whole organisms due to lack of sensitivity", stated the document.[91]

On 20 November, Anne Glover did inform DG Environment, DG SANCO and the Secretariat General about the outcome of the scientific meeting. One might think that such a spectacular U-turn, putting an end to the 'controversy', would shatter the Commission and annihilate its convenient excuse to make an impact assessment. But it didn't.

By December 2013, the Commission had missed the deadline officially set by the 2009 pesticide regulation, nor did it schedule a new one.

MEPs of the Socialist Group (S&D) reminded the Commission that it had missed the legal deadline and should have already published the scientific criteria for EDCs.[92]

On 25 March 2014, the group of eight MEPs finally received a reply to their October 2013 letter to Barroso. Signed by Karl Falkenberg (DG Environment) and Paola Testori Coggi (DG SANCO), it justified the impact assessment not only for "concerns about the possible potential significant impacts on some sectors" associated with any set of EDC criteria, but also "the vigorous debate in the scientific community on endocrine disruptors that escalated over last summer".[93] Again, the Commission chose to ignore the fact that this debate had been extinguished in Anne Glover's office months earlier.

And it was not only in the Parliament that people were very upset. In March 2014, Sweden decided to bring the Commission to court for "failure to act".

A Roadmap to nowhere

Now industry's attention shifted to the terms of the impact assessment. DG Environment and SANCO were charged with designing a 'Roadmap' that would set out the scope of the impact assessment, and that would present the policy options to be assessed within it. They convened a first meeting of the Impact Assessment Steering Group on 20 January 2014, inviting participants from across the Commission, including Research, Climate, Agriculture, Enterprise, and Trade.[94]

The pesticide lobby ECPA was clearly aware of this meeting, as well as who would be there. One week before, on 13 January, they sent their "suggestions" on the impact assessment to DG SANCO.[95] A few days later, CEFIC did the same.[96]

ECPA and CEFIC obviously made almost identical points. Among other things, they demanded that in order to arrive at a "meaningful assessment" of the impacts of EDC criteria, the impact assessment should be "sufficiently

detailed", ie spelling out the expected impacts separately for each pesticide or group of pesticides, and for their specific uses. But since this would give a clear indication to the outside world which pesticides the Commission suspected to be endocrine disruptors, they "strongly recommended" to leave the public in the dark. In their own words: "this should not be published in a way that creates a public list of suspected endocrine disruptors: past experience has shown that some stakeholders may use such a list as a 'black list' thereby introducing the potential for unfair competition".

On 20 June 2014, after months of tough negotiations between DG Environment and DG SANCO, the Roadmap was finally published.[97] Surprise! The potency criterion was back on the table, despite the fact that it had been ruled out by DG Environment's expert group in March 2013.[98] The next step was a public consultation on this flawed Roadmap, launched on 29 September 2014.[99]

Box 12 The US government submission to the public consultation steps up the level of bluntness by dismissing DG Environment's proposal as "a failure to adopt a scientific approach", that could impact – quoting corporate estimates – €65.3 billion worth of imports into the EU (of which over €4 billion worth would be US exports).[100]

Juncker's removal company

Meanwhile, the European elections of May 2014 had led to a new Parliament and a new team of Commissioners. On 10 September 2014, the new President of the European Commission Jean-Claude Juncker announced the names of the new Commissioners and his priorities for the next five years. At the bottom of the press release, there was a long table detailing the changes of tasks within the Commission.[101] DG Environment was being officially removed from their leading role on the EDC criteria which would now become the responsibility of... DG SANCO.

The impact assessment on the EDC criteria will still take a long time to be completed. Even in the best-case scenario, the EDC criteria will not be ready before the second half of 2016. The chemical and pesticide industry lobbying will continue unabated along the two parallel tracks: the obstructed EU process (see Box 13), and the TTIP negotiations.

Adding more voices to the chorus

Box 13

More examples of the industry lobbying trick to mobilise 'third voices' were seen in Brussels. In January 2015 BASF sponsored a 'Science Policy Breakfast meeting' on EDCs[102] with a presentation by Richard Sharpe, a professor of Reproductive health at the University of Edinburgh and one of the signatories to the letter to Anne Glover. The event was hosted by MEP Jan Huitema (Dutch Liberals). Richard Sharpe said that unlike Kortenkamp he was not a "believer" in EDCs having no safe threshold. In February the British National Farmers Union (NFU, member of COPA-COGECA), in tandem with the British pesticide industry brought their "Healthy Harvest campaign" to Brussels.[103] The core of this campaign "for sound, science-based regulation" of pesticides was a report commissioned from a UK consultancy Andersons, concluding that the impact on UK agriculture would be "severe", if pesticides would be removed from the market as a result of "overly precautionary definitions of endocrine disruptors". In Brussels, ECPA and COPA-COGECA have joint events in the European Parliament. Two ECPA lobbyists were recruited from the NFU and a Polish member organisation of COPA-COGECA.

But there are also glimmers of hope in both tracks. In an unprecedented move, in January 2015, both the European Parliament *and* the Council (all Member States together) decided to officially support Sweden's court case against the Commission over its failure to establish criteria for EDCs.[104] An overwhelming 21 Member States voted in favour, while only a few abstained, such as the UK. The TTIP negotiations are troubled by increasing critical public debate and resistance. The battle around this key public health and environment policy in the EU is far from over.

Annex I

March-July 2013. Examples of industry lobby communications to the European Commission on EDC criteria, nearly all calling for impact assessment.

Date	What	Sender	Target
08 March	Email including ECPA's own impact assessment	ECPA	DG Environment, SANCO, Enterprise, Trade, and the Joint Research Center
11 March	Email including ECPA's own impact assessment	ECPA	Janez Potočnik, Environment Commissioner
14 March	Email including ECPA's own impact assessment	BASF	DG Enterprise
23 March	Email including ECPA's own impact assessment	ECPA	Secretariat General
16 April	Email including ECPA's own impact assessment	?	DG Enterprise
21 May	Letter	COPA-COGECA	DG Environment
29 May	Request meeting	AmCham and EPPA	Anne Glover
06 June *	Letter	CEFIC	Bjorn Hansen DG Environment
07 June	Email including ECPA's own impact assessment	Bayer	Marianne Klingbeil in the Secretariat General
07 June	Email	AmCham	DG Enterprise
13 June	Meeting	Bayer	DG SANCO
19 June *	Meeting and follow up mail	ECPA	DG Agriculture
20 June *	Meeting	ECPA	Duncan Johnstone and Stefan Fuering in the Secretariat General
21 June	Email including ECPA's own impact assessment (Three industry-commissioned impact assessments in attachment)	ECPA	DG Enterprise
21 June *	Meeting	ECPA and BASF	Fabrizia Benini, member of cabinet of Antonio TAJANI, Enterprise Commissioner
24 June	Letter	CEFIC Director General Hubert Mandery	Janez Potočnik, Environment Commissioner
25 June	Email	CEFIC	DG Enterprise
25 June	Follow-up email	ECPA	Fabrizia Benini, member of cabinet of Vice-President Antonio Tajani, Enterprise Commissioner
26 June	Meeting on TTIP	AmCham EU	DG Enterprise and Trade
?	Email including ECPA's own impact assessment	?	DG Trade
27 June	Email including ECPA's own impact assessment	ECPA	Duncan Johnstone and Stefan Fuering in the Secretariat General
11 July	Email including ECPA's own impact assessment	Bayer	SANCO

* = not or unclear if discussing the impact of the criteria or requesting an impact assessment.

Emails are available from Corporate Europe Observatory and Stéphane Horel.

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STEPHANE HOREL

From: Pete Myers <jpmyers@ehsic.org>
To: "Karp, Harvey" <montee@earthlink.net>, "Prins, Gail" <gprins@uic.edu>, "Lanphear, Bruce" <blanphear@sfu.ca>, "Cranmer, Joan" <cranmerJoanM@uams.edu>, "Cory-Slechta, Deborah" <deborah_cory-slechta@urmc.rochester.edu>, Peter Orris <porris@uic.edu>, "Prof. Fred vom Saal" <vomsaalf@missouri.edu>, Terry Collins <tc1u@andrew.cmu.edu>, Howard Snyder <snyderh@email.chop.edu>, Peter DeFur <pldefur@igc.org>, "Ho, Shuk-mei" <shuk-mei.ho@uc.edu>, "Zoeller, Tom" <tzoeller@bio.umass.edu>, "Prof. Louis J. Guillette" <lou.guillette@gmail.com>, Ted Schettler <tschettler@igc.org>, "Ozonoff, David" <dozonoff@bu.edu>, "Hayes, Tyrone" <tyrone@berkeley.edu>, "Woodruff, Tracey" <WoodruffT@obgyn.ucsf.edu>, "Dr. Steve Heilig" <heilig@sfms.org>, "Stahlhut, Richard" <richard_stahlhut@urmc.rochester.edu>, Sheldon Krimsky <sheldon.krimsky@tufts.edu>, Philip Landrigan <phil.landrigan@mssm.edu>, "Hunt, Pat" <pathunt@wsu.edu>, Shanna Swan <shanna.swan@mssm.edu>, "Hauser, Russ" <rhauser@hohp.harvard.edu>, Bruce Blumberg <blumberg@uci.edu>, "Amy Kostant" <amy@sciencecom.org>, Bernard Weiss <Bernard_Weiss@urmc.rochester.edu>, Kalee Kreider <kaleekreider@gmail.com>, Laura Vandenberg <lvandenberg@schoolph.umass.edu>, Emily Copeland <emily@sciencecom.org>, Carl-Gustaf Bornehag <caguborn@kau.se>, "Michael Antoniou" <michael.antoniou@kcl.ac.uk>, Steve Gilbert <sgilbert@innd.org>, Leonardo Trasande <leonardo.trasande@nyu.edu>, Amy Itescu <itescua@UCMAIL.UC.EDU>
Sent: 3/25/2015 6:29:14 AM
Subject: Recipe for failing an experiment

Attach: [2015-0325 recipe.docx]

Fred vS and I spent the day yesterday with an investigative reporter talking about various dimensions of the FDA and BPA. One of the things we did was deconstruct the FDA¹s shenanigans with CLARITY, the collaborative experiment involving FDA, NIEHS and 14 PIs involved in CLARITY.

We took the many missteps that FDA has committed and then generalized them in the attached list of ³things to do if you want an experiment to fail.²

Perhaps we can talk about this today, or not. The agenda is packed. But I would appreciate feedback on this list as well as additional tricks.¹ Perhaps it would be useful to develop a parallel list for epidemiological studies.

Not sure what we¹re going to do with this. But the exercise has been useful.

- Recipe for causing an experiment to fail
  - Making data go away
    - inappropriate statistics
    - conflict between data and conclusions/abstract
  - Taking steps to undermine experiment
    - Wrong animals chosen for study
    - Wrong delivery mechanism (e.g., gavage)
    - Contaminated controls
    - Contaminated assays
    - Increase variance
    - Decrease sample size to reduce power
    - Sloppy methodology
    - Mess up samples (e.g., smooshed brains)
  - Slow/stop publication (while FDA continues to publish junk)
  - Censor publication content
  - Declare nonmonotonicity “no dose response”

From: "Hunt, Pat" <pathunt@vetmed.wsu.edu>
To: Pete Myers <jpmyers@ehsic.org>
Sent: 11/8/2016 7:46:12 AM
Subject: reference

Hi Pete-

I hope all is well in your new world. I am on sabbatical at The Jackson Laboratory in Maine for the semester. This is the first sabbatical my husband and I have ever taken, and we are kicking ourselves for not doing it sooner. We are mostly writing papers but also attending lab meetings and interacting with colleagues here. The chance to step outside ones life provides a great opportunity to consider the future which leads me to my reason for writing. I am considering a major career change and submitting applications for a couple of administrative positions. I am writing to ask if I can list you as a reference.

The positions are a bit of a long shot, so likely you will not have to do a thing. However, I must admit that the thought of using what I have learned to motivate young people has my juices flowing.

With warm regards,

Pat

Patricia Hunt
Meyer Distinguished Professor
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PO Box 647520
Pullman, WA 99164-7520 (for letters)
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509-335-4954

From: Pete Myers <jpmyers@ehsic.org>
To: Sonya Lunder <sonya@ewg.org>
Sent: 3/17/2015 7:02:54 AM
Subject: Reforming the Toxic Substance Control Act

Attach: [2015-TSCA-scientists letter-clean.docx]

Apologies for cross-posting.

The Environmental Working Group is submitting a letter, attached, to Senators James Inhofe and Barbara Boxer about the Udall-Vitter bill that has been proposed as a replacement. They are asking for scientists who agree with the issues they raise to email Sonya Lunder at EWG their willingness to be a signatory to the letter, if that is the case. sonya@ewg.org

[Date]

Senator James Inhofe
Chairman, Environment & Public Works Committee
410 Dirksen Senate Office Building
Washington, D.C. 20510

Senator Barbara Boxer
Ranking Member, Environment & Public Works Committee
456 Dirksen Senate Office Building
Washington, D.C. 20510

Dear Chairman Inhofe and Ranking Member Boxer:

We the undersigned scientists thank you for your interest in reforming the Toxic Substances Control Act to achieve its original purposes. The Toxic Substances Control Act, passed in 1976 with bipartisan support, was intended to create a system to protect the public from the effects of harmful chemicals. It is widely acknowledged to have failed and to require an overhaul. We are concerned that the current bill to reform TSCA (S. 697) does not adequately address the most important deficiencies in U.S. chemical regulation.

Since World War II, chemical production and use in the U.S. has increased dramatically. There are currently more than 80,000 chemical substances registered for use in U.S. commerce; several thousand of them are manufactured or imported in excess of 1 million pounds each every year.

We have good reasons to be concerned about widespread human exposure to chemicals in use. Scientists have developed the ability to detect trace chemicals in the human body and have shown that many Americans are exposed daily to dozens of chemicals linked to potentially harmful health effects. The federal Centers for Disease Control and Prevention sponsors the National Health and Nutrition Examination Survey.[1] It is a nationally representative study of the population that measures chemicals in blood and urine and consistently finds hundreds of chemicals in Americans, particularly in pregnant women and children.[2]

Dozens of pollutants are now known to cross the human placenta from mother to child during pregnancy, some at concentrations known to adversely affect neurological and reproductive systems. It is also clear that subtle damages to individual children can result in major consequences at the population level. One study has estimated that three common pollutants alone – mercury, lead and organophosphate pesticides – result in a total decrease of 40 million IQ points among American children ages 0 to 5 years as a group.[3] Laboratory and observational studies suggest that scores of other widely used chemicals may also cause toxic effects, but in

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[1] Centers for Disease Control. 2013. Report on Human Exposure to Environmental Chemicals. National Health and Nutrition Examination Survey, http://www.cdc.gov/exposurereport/.

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many cases the extent of the risks to children has not been adequately determined.

Many of the chemicals detected in people's bodies are released from industrial sites into air and water and ultimately reach the food supply. Thousands more are intentionally added to consumer products. While regulatory programs for air and water have made improvements, TSCA has failed to protect Americans from chemicals. Modeling suggests that chemicals used in household consumer products can pose a more direct exposure risk for the general population than chemicals with only industrial uses.[4]

Many chemicals have not been adequately studied for their effects on human health, primarily because TSCA does not require manufacturers to ensure the safety of the chemicals they produce. The U.S. Environmental Protection Agency (EPA) has insufficient authority to require health and safety data, and insufficient resources to conduct the testing itself.

EPA leadership recently declared that "absent statutory changes, the Agency will not be able to successfully meet the goal of ensuring chemical safety now or in the future."[5] As scientists, we urge you to create a modern and robust system that will allow EPA to fully assess chemical hazards and protect public health. A reformed TSCA should also be harmonized with chemical regulations in Europe, Japan, Canada, and Australia; the U.S. does not need to reinvent the wheel.

Specifically, we urge you to address the following principles in any effort to update the Toxic Substances Control Act:

• Congress must authorize EPA to restrict chemicals that threaten human health

EPA is currently only authorized to address the "unreasonable risks" that chemicals pose to human health or the environment. When a chemical fails even this weak safety standard, the agency faces unreasonably burdensome requirements when it attempts to restrict the chemical's use. Efforts to improve TSCA must provide a greater degree of public health protection, using the truly health-protective safety standard "reasonable certainty of no harm," and must remove unwarranted procedural obstacles to EPA's ability to address chemicals that fail to meet the standard.

• EPA must comply with modern scientific principles in its assessments of chemical risks.

EPA risk assessments should be required to conform to the recommendations of the National Academy of Sciences (NAS).[6] The NAS has urged EPA to better assess the scientific weight of evidence on chemical toxicity, and characterize data gaps and uncertainty around its decisions. EPA must assess human variability in both exposure to chemicals and sensitivity to toxic effects. This should explicitly include the aggregate effects of chemical mixtures.[7] Furthermore NAS

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[4] Wambaugh JF, et al. 2013. High-Throughput Models for Exposure-Based Chemical Prioritization in the ExpoCast Project. Environmental Science and Technology. 47:8479-88.

[5] GAO. 2013. Toxic Substances: EPA has increased efforts to assess and control chemicals but could strengthen its approach. U.S. Government Accountability Office. GAO-13-249.

has called for a unified approach to considering cancer and non-cancer hazards. This would mean that EPA should assume that low levels of exposure to chemicals are associated with some level of risk unless sufficient data is available to contradict this assumption.[8] Reform of TSCA must make EPA compliance with the recommendations of the NAS mandatory.

• EPA must move quickly to screen new and existing chemicals

EPA's previous efforts to evaluate the hazards posed by high production volume chemicals, potential endocrine disruptors, or, most recently, to assess risks of 83 high priority chemicals, have been subject to numerous delays. Without clear authority, statutory deadlines and funding, EPA scientists will be unable to efficiently review and assess the thousands of industrial chemicals produced in high volumes. For example, the U.S. Government Accountability Office (GAO) found that, at the current pace, it could take EPA at least 10 years to assess just 83 chemicals of high concern, not to mention hundreds of other widely used and poorly studied chemicals.[9] Any new legislation must include clear and swift deadlines for the prioritization, assessment, and regulation of chemicals of concern. EPA must also be equipped the with the financial and personnel resources necessary to conduct chemical safety evaluations adequately and in a timely manner.

• Scientists are willing to help

As scientists and public health professionals, we have dedicated our professional lives to better understanding chemicals' effects on human health and the environment. On the basis of this research we conclude that TSCA must be reformed to provide EPA with the authority it needs to fill data gaps and to restrict chemicals that pose clear risks to people and the environment. The scientific community has valuable expertise and must be at the table as TSCA is rewritten. With scientific input, we can learn from past mistakes and benefit from decades of research on chemicals' environmental fates and effects. Only then will we collectively be able to protect public health from these chemical hazards.

Sincerely,

[New signers]

[9] GAO. 2013. Toxic Substances: EPA has increased efforts to assess and control chemicals but could strengthen its approach. U.S. Government Accountability Office. GAO-13-249.

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 1/27/2016 6:21:33 AM
Subject: Remdinder: SCN New Science Call today, 1/27, noon eastern

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Hi all -

This is a reminder that the next SCN new science call will be held today, January 27, at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: 41

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org

From: Emily Copeland
Sent: Wednesday, January 20, 2016 10:33 AM
To: 'tc1u@andrew.cmu.edu'; 'CranmerJoanM@uams.edu'; 'deborah_cory-slechta@urmc.rochester.edu'; 'pldefur@igc.org'; 'sgilbert@innd.org'; 'tyrone@berkeley.edu'; 'heilig@sfms.org'; 'pathunt@wsu.edu'; 'dickjackson@ucla.edu'; 'dr.karp@thehappiestbaby.com'; 'phil.landrigan@mssm.edu'; 'BLanphear@sfu.ca'; 'JPMyers@ehsic.org'; 'hlnlead@pitt.edu'; 'porris@uic.edu'; 'dozonoff@bu.edu'; 'gprins@uic.edu'; 'tschettler@igc.org'; 'snyderh@email.chop.edu'; 'shanna.swan@mssm.edu'; 'vomsaalF@missouri.edu'; 'bernard_weiss@urmc.rochester.edu'; 'WoodruffT@obgyn.ucsf.edu'; 'tzoeller@bio.umass.edu'; 'shuk-mei.ho@uc.edu'; 'stahlhutr@missouri.edu'; 'blumberg@uci.edu'; 'svogel@edf.org'; 'lvandenberg@schoolph.umass.edu'; 'carl-gustaf.bornehag@kau.se'; 'RHAUSER@hohp.harvard.edu'; 'leonardo.trasande@nyu.edu'; 'kkreider@unfoundation.org'; 'itescua@UCMAIL.UC.EDU'; 'michael.antoniou@kcl.ac.uk'; sheldon.krimsky@tufts.edu
Cc: Amy Kostant - Science Communication Network (amy@sciencecom.org); Gabriela Silvani (gabriela@sciencecom.org)
Subject: SCN New Science Call next Wednesday, 1/27, noon

Hi all -
This is a reminder that this month's SCN new science call will be held next **Wednesday, January 27th at noon eastern**. If you'd like to present new or soon-to-be released science, discuss a particular topic or share key points from a recent conference, please let me know so I can include it on the agenda.

****Please note the call-in information:**
Dial in: 1-888-537-7715
Code: 41

Thanks in advance to those of you who let me know if you plan to join the call.

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org
@EmilySCN

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>, "jgallen@hsph.harvard.edu" <jgallen@hsph.harvard.edu>, "andreas.kortenkamp@brunel.ac.uk" <andreas.kortenkamp@brunel.ac.uk>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 6/30/2016 12:08:28 PM
Subject: Reminder: SCN New Science Call next Wednesday, July 6th at noon eastern

---

Hi All -

This is a reminder that this month's SCN new science call

will be held next

Wednesday, April at noon

eastern

. If you'd like to present new or soon-

to

-be released science, discuss a particular topic or share

key

points from a recent conference, please let me know so

I can include it on the agenda.

** Please note the call-in information:

Dial in: 1-888-537-7715

Code: [redacted]

Thanks in advance to those of you who let me know if you pl

an to join the call.

Best,

Emily

Hi All -

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Wednesday, April at noon

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Code: [redacted]

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Code: [redacted]

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**** Please note the call-in information:**

Dial in: 1-888-537-7715

Code: [redacted]

Thanks in advance to those of you who let me know if you plan to join the call.

Best,

Emily

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>, "jgallen@hsph.harvard.edu" <jgallen@hsph.harvard.edu>, "andreas.kortenkamp@brunel.ac.uk" <andreas.kortenkamp@brunel.ac.uk>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 8/31/2016 6:35:46 AM
Subject: Reminder: SCN New Science call today (8/31) at noon eastern

---

Hi all -

This is a reminder that the next SCN new science call will be held today at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion
    - NIEHS 25th Anniversary Meeting in Bethesda (9/18 9/20)

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: [redacted]

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org
@EmilySCN

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>, "jgallen@hsph.harvard.edu" <jgallen@hsph.harvard.edu>, "andreas.kortenkamp@brunel.ac.uk" <andreas.kortenkamp@brunel.ac.uk>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 9/28/2016 5:44:18 AM
Subject: Reminder: SCN New Science Call today at noon eastern

---

Hi all -
This is a reminder that the next SCN new science call will be held today at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: [redacted]

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org

From: Emily Copeland
Sent: Monday, September 26, 2016 10:36 AM
To: 'tc1u@andrew.cmu.edu'; 'CranmerJoanM@uams.edu'; 'deborah_cory-slechta@urmc.rochester.edu'; 'pldefur@igc.org'; 'sgilbert@innd.org'; 'tyrone@berkeley.edu'; 'heilig@sfms.org'; 'pathunt@wsu.edu'; 'dickjackson@ucla.edu'; 'dr.karp@thehappiestbaby.com'; 'phil.landrigan@mssm.edu'; 'BLanphear@sfu.ca'; 'JPMyers@ehsic.org'; 'hlnlead@pitt.edu'; 'porris@uic.edu'; 'dozonoff@bu.edu'; 'gprins@uic.edu'; 'tschettler@igc.org'; 'snyderh@email.chop.edu'; 'shanna.swan@mssm.edu'; 'vomsaalF@missouri.edu'; 'bernard_weiss@urmc.rochester.edu'; 'WoodruffT@obgyn.ucsf.edu'; 'tzoeller@bio.umass.edu'; 'shuk-mei.ho@uc.edu'; 'stahlhutr@missouri.edu'; 'blumberg@uci.edu'; 'svogel@edf.org'; 'lvandenberg@schoolph.umass.edu'; 'carl-gustaf.bornehag@kau.se'; 'RHAUSER@hohp.harvard.edu'; 'leonardo.trasande@nyu.edu'; 'kkreider@unfoundation.org'; 'itescua@UCMAIL.UC.EDU'; 'michael.antoniou@kcl.ac.uk'; 'sheldon.krimsky@tufts.edu'; 'jgallen@hsph.harvard.edu'; andreas.kortenkamp@brunel.ac.uk
Cc: Amy Kostant - Science Communication Network (amy@sciencecom.org); Gabriela Silvani (gabriela@sciencecom.org)
Subject: SCN New Science Call next Wednesday, 9/28, noon

Hi all -
This is a reminder that this month's SCN new science call will be held next **Wednesday, September 28th at noon eastern**. If you'd like to present new or soon-to-be released science, discuss a particular topic or share key points from a recent conference, please let me know so I can include it on the agenda.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: [redacted]

Thanks in advance to those of you who let me know if you plan to join the call.

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org
@EmilySCN

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>, "jgallen@hsph.harvard.edu" <jgallen@hsph.harvard.edu>, "andreas.kortenkamp@brunel.ac.uk" <andreas.kortenkamp@brunel.ac.uk>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 7/6/2016 7:35:58 AM
Subject: Reminder: SCN New Science Call today at noon eastern

---

Hi all,
This is a reminder that the next SCN new science call will be held today, July 6th, at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion

If you'd like to add anything to this agenda, please let me know.

*Please note the call-in information:
Dial in: 1-888-537-7715
Code: [redacted]

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,

Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org

From: Emily Copeland
Sent: Thursday, June 30, 2016 3:08 PM
To: tc1u@andrew.cmu.edu; CranmerJoanM@uams.edu; deborah_cory-slechta@urmc.rochester.edu; pldefur@igc.org; sgilbert@innd.org; tyrone@berkeley.edu; heilig@sfms.org; pathunt@wsu.edu; dickjackson@ucla.edu; dr.karp@thehappiestbaby.com; phil.landrigan@mssm.edu; BLanphear@sfu.ca; JPMyers@ehsic.org; hlnlead@pitt.edu; porris@uic.edu; dozonoff@bu.edu; gprins@uic.edu; tschettler@igc.org; snyderh@email.chop.edu; shanna.swan@mssm.edu; vomsaalF@missouri.edu; bernard_weiss@urmc.rochester.edu; WoodruffT@obgyn.ucsf.edu; tzoeller@bio.umass.edu; shuk-mei.ho@uc.edu; stahlhutr@missouri.edu; blumberg@uci.edu; svogel@edf.org; lvandenberg@schoolph.umass.edu; carl-gustaf.bornehag@kau.se; RHAUSER@hohp.harvard.edu; leonardo.trasande@nyu.edu; kkreider@unfoundation.org; itescua@UCMAIL.UC.EDU; michael.antoniou@kcl.ac.uk; sheldon.krimsky@tufts.edu; jgallen@hsph.harvard.edu; andreas.kortenkamp@brunel.ac.uk
Cc: Amy Kostant; Gabriela Silvani
Subject: Reminder: SCN New Science Call next Wednesday, July 6th at noon eastern

Hi All -

This is a reminder that this month's SCN new science call

will be held next

Wednesday, April at noon

eastern

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key

points from a recent conference, please let me know so

I can include it on the agenda.

** Please note the call-in information:

Dial in: 1-888-537-7715

Code: [redacted]41

Thanks in advance to those of you who let me know if you pl

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Best,

Emily

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Dial in: 1-888-537-7715

Code: [redacted]

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Dial in: 1-888-537-7715

Code: [redacted]

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Emily

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**** Please note the call-in information:**

Dial in: 1-888-537-7715

Code: [redacted]

Thanks in advance to those of you who let me know if you plan to join the call.

Best,

Emily

From: Emily Copeland <emily@sciencecom.org>
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CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 11/30/2016 7:11:38 AM
Subject: Reminder: SCN New Science Call today noon eastern

Hi all -
This is a reminder that the next SCN new science call will be held today at noon eastern. The proposed agenda for the call is:

AGENDA:
- Call for new science; updates on conferences; etc.
- "Dear Journalist" notes and media response
- Discussion
- Welcome new Board member: Chris Portier
- Sperm crisis in China
- Science integrity

If you'd like to add anything to this agenda, please let me know.

*Please note the call-in information:
Dial in: 1-888-537-7715

Code: [redacted]

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
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-----Original Message-----
From: Emily Copeland
Sent: Monday, November 28, 2016 11:57 AM
To: tc1u@andrew.cmu.edu; CranmerJoanM@uams.edu; deborah_cory-slechta@urmc.rochester.edu; pldefur@igc.org; sgilbert@innd.org; tyrone@berkeley.edu; heilig@sfms.org; pathunt@wsu.edu; dickjackson@ucla.edu; dr.karp@thehappiestbaby.com; phil.landrigan@mssm.edu; BLanphear@sfu.ca; JPMyers@ehsic.org; hlnlead@pitt.edu; porris@uic.edu; dozonoff@bu.edu; gprins@uic.edu; tschettler@igc.org; snyderh@email.chop.edu; shanna.swan@mssm.edu; vomsaalF@missouri.edu; bernard_weiss@urmc.rochester.edu; WoodruffT@obgyn.ucsf.edu; tzoeller@bio.umass.edu; shuk-mei.ho@uc.edu; stahlhutr@missouri.edu; blumberg@uci.edu; svogel@edf.org; lvandenberg@schoolph.umass.edu; carl-gustaf.bornehag@kau.se; RHAUSER@hohp.harvard.edu; leonardo.trasande@nyu.edu; kkreider@unfoundation.org; itescua@UCMAIL.UC.EDU; michael.antoniou@kcl.ac.uk; sheldon.krimsky@tufts.edu; jgallen@hsph.harvard.edu; andreas.kortenkamp@brunel.ac.uk; Chris Portier
Cc: Amy Kostant; Gabriela Silvani
Subject: SCN New Science Call this Wednesday, 11/28, noon eastern

Hi all -
This is a reminder that this month's SCN new science call will be held this Wednesday, November 28th at noon eastern. If you'd like to present new or soon-to-be released science, discuss a particular topic or share key points from a recent conference, please let me know so I may include it on the agenda.

*Please note the call-in information:
Dial in: 1-888-537-7715
Code: [redacted]

Thanks in advance to those of you who let me know if you plan to join the call.

Best,

Emily

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>, "jgallen@hsph.harvard.edu" <jgallen@hsph.harvard.edu>, "andreas.kortenkamp@brunel.ac.uk" <andreas.kortenkamp@brunel.ac.uk>
CC: Amy Kostant <amy@sciencecom.org>
Sent: 7/27/2016 8:19:34 AM
Subject: Reminder: SCN New Science call today noon eastern

Attach: [Eckelman and Sherman Env Impacts of US Health Care 2016.pdf]
Hi all -
This is a reminder that the next SCN new science call will be held today at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion
    - **12:15 -12:35** Peter Orriss guest introduction: Jodi Sherman (anesthesiologist) from Yale; and Matthew Eckleman from Northeastern University College of Engineering

Thanks to Peter Orris, we are pleased to have Jodi Sherman from the Department of Anesthesiology at the Yale School of Medicine and Matthew Eckelman from Northeastern University of Engineering, join the call to discuss their paper on healthcare system impacts on public health. Their paper, Environmental Impacts of the U.S. Health Care System and Effects on Public Health published in *PLOS ONE*, and is attached here for reference.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: [redacted]

Thanks to all who have RSVP'd. Looking forward to speaking with you soon,

Best,
Emily

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PLOS ONE

RESEARCH ARTICLE

Environmental Impacts of the U.S. Health Care System and Effects on Public Health

Matthew J. Eckelman[1]*, Jodi Sherman[2]

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OPEN ACCESS

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Competing Interests: The authors have declared that no competing interests exist.

Abstract

The U.S. health care sector is highly interconnected with industrial activities that emit much of the nation's pollution to air, water, and soils. We estimate emissions directly and indirectly attributable to the health care sector, and potential harmful effects on public health. Negative environmental and public health outcomes were estimated through economic input-output life cycle assessment (EIOLCA) modeling using National Health Expenditures (NHE) for the decade 2003–2013 and compared to national totals. In 2013, the health care sector was also responsible for significant fractions of national air pollution emissions and impacts, including acid rain (12%), greenhouse gas emissions (10%), smog formation (10%) criteria air pollutants (9%), stratospheric ozone depletion (1%), and carcinogenic and non-carcinogenic air toxics (1–2%). The largest contributors to impacts are discussed from both the supply side (EIOLCA economic sectors) and demand side (NHE categories), as are trends over the study period. Health damages from these pollutants are estimated at 470,000 DALYs lost from pollution-related disease, or 405,000 DALYs when adjusted for recent shifts in power generation sector emissions. These indirect health burdens are commensurate with the 44,000–98,000 people who die in hospitals each year in the U.S. as a result of preventable medical errors, but are currently not attributed to our health system. Concerted efforts to improve environmental performance of health care could reduce expenditures directly through waste reduction and energy savings, and indirectly through reducing pollution burden on public health, and ought to be included in efforts to improve health care quality and safety.

Introduction

The Institute of Medicine 2013 Workshop Summary *Public Health Linkages with Sustainability* suggests that "the health sector should lead by example by greening itself and reducing its ecological footprint. . ..to improve global health and the health of the planet [1]." Quantification of pollution and disease burden stemming from health care is critical to improve the quality and safety of practice, to inform mitigation strategies and leverage health care leadership in sustainable development.

The United States spends the most of any nation by far on its health care system, nearly one-fifth of GDP, or approximately $3 trillion dollars in 2013 [2]. Health care services are also energy-intensive. Hospitals are the second-most energy-intensive commercial buildings in the country, after food service facilities [3]. Hospitals are typically large buildings, open 24 hours a day, seven days a week, and contain several energy-intensive activities, including sophisticated heating, cooling, and ventilation systems, computing, medical and laboratory equipment use, sterilization, refrigeration, laundry, as well as food service [3]. In addition to energy used on site in the form of heating fuels and electricity, the health care system also uses vast quantities of energy-intensive goods and services, such as pharmaceuticals and medical devices, which require significant energy inputs for their manufacturing. As the U.S. is the second-largest emitter of greenhouse gases (GHGs) globally, it follows that the health care sector is an important target for emissions reductions as well. Yet despite its size and status, there has been little work to quantify or probe consumption-based emissions from the U.S. health care sector, how these emissions are trending over time, or how these emissions might affect public health overall.

In 2009, Chung and Meltzer [4] estimated the aggregate carbon footprint of the U.S. health care sector, underscoring the substantial role that health care plays in the physical economy of the country. They report that health care contributes 8% of the nation's greenhouse gas (GHG) emissions both from health care activities and direct purchases (46%) and from indirect activities associated with the supply chain of health care-related goods and services (54%). Parallel efforts in the United Kingdom report that the National Health Service (NHS) contributes 3–4% of the national GHG emissions total [5]. Efforts are underway to quantify the climate impacts of specific medical devices or supplies and procedures, with the overall objective of finding equally effective but less carbon-intensive ways to deliver care [6–9].

While greenhouse gases (GHGs) are a critical category of emissions and climate change may have severe, negative impacts on human health and livelihoods [10], there are several other categories of emissions from health care with negative environmental and public health consequences that are important to consider. In addition to direct emissions from health care facilities, there are also indirect emissions that occur as a consequence of producing the electricity and materials that those facilities use. In this way, the health care sector is interconnected with and supported by industrial activities that emit much of the pollution to air, water, and soils nationally, including particulate matter, sulfur and nitrogen oxides, persistent organic pollutants, and toxic metals. These very emissions contribute to the national disease burden. Fine particulate matter is the leading cause of air pollution-related disease, with 87% of the world's population living in areas exceeding the World Health Organization (WHO) Air Quality Guideline of 10 $\mu g/m^3$ $PM_{2.5}$ [11]. The objective of this work is to provide a quantitative estimate of the GHG and non-GHG-related emissions directly and indirectly attributable to the U. S. health care sector in order to assess the scale of potential harmful effects of these emissions on public health.

Methods

Negative environmental and public health outcomes attributable to the health care sector were estimated for the U.S. using economic input-output life cycle assessment (EIOLCA). Input-output models are compiled by the federal Bureau of Economic Analysis (BEA) and describe monetary flows among all of the 400+ economic sectors that comprise the national economy. EIOLCA extends these economic models by adjoining sector-specific intensity values for emissions and resource use (*e.g.*, energy, water) per dollar of expenditure. As described in the model documentation [12], for those sectors that report energy use or emissions data, such as utilities

or manufacturing, intensity values come from government agencies such as the Environmental Protection Agency (EPA) or Energy Information Administration (EIA). For other EIOLCA sectors for which resource use and emissions are not reported directly, researchers extracted economic data from the BEA Commodity x Industry Use table to determine total expenditures on different fuel commodities (*e.g.*, coal, oil, natural gas), used average price data to translate these dollar values to physical amounts of fuels, and used emission factors for each fuel to estimate total emissions of different pollutants. Once all intensity values have been adjoined, matrix algebra model algorithms then use economic activity in a given sector to calculate both direct emissions (from that sector) and indirect emissions (from all other linked sectors) that occur throughout the entire economy as a result of that activity [13].

We use health care spending data compiled in the US National Health Expenditure Accounts for the decade 2003–2013 in all categories of health consumption and investment expenditures [2]. Each expenditure category is matched to the corresponding economic sector (S1 Table) in the most recent 2002-vintage purchaser-price EIOLCA model, housed at Carnegie Mellon University [12]. This model requires inputs in nominal 2002 dollars, so expenditures in subsequent years are deflated to this base year using the National Health Expenditures Medical Price Index [2], calculated using the component-based Producer Price Index and Consumer Price Index from the Bureau of Labor Statistics (S2 Table). This approach allows for a dynamic view of health care-related emissions and damages over the study period.

For each expenditure, the EIOLCA model then returns direct and indirect emissions to air, water and soils; these emissions form the life cycle inventory (LCI) of the results. These emissions are then linked to nine categories of environmental and human health outcomes, included in this analysis in order to quantify the contribution of health care-related activities relative to national totals. These impact categories include global warming; stratospheric ozone depletion (allowing higher levels of short-wave ultraviolet light through the atmosphere, increasing the health risks of skin cancer); respiratory disease from inhalation of primary and secondary particulate matter (PM) and from ground-level ozone (smog) stemming from emissions of criteria air pollutants; cancer and non-cancer disease through inhalation and ingestion routes of chemical exposure; environmental effects of acidification (from formation and deposition of acid rain) and eutrophication (algae blooms from excess nutrients) in soils and surface waters; and ecotoxicity that reflects the toxic burden of all emitted chemicals to aquatic organisms. Emissions are linked from the EIOLCA model to these nine categories of environmental and human health impacts using the USEPA's life cycle chemical fate-exposure-effect model (TRACI) [14]. Each emitted substance that contributes to a particular environmental or health impact is then scaled by its impact-specific potency, represented by a 'characterization factor'. Each factor is endpoint- and substance-specific and is a complex function of a chemical's fate and transformation in the environment, chemical activity, uptake and exposure, and potential toxicity. In the public health context, characterization factors measure average health damages per unit of chemical emitted. Characterization factors are relative, measured against a reference substance for which effects are well-known, and so have common equivalent units, such as CO_2-e for global warming [15].

In life cycle assessment methodology (ISO 14040/44), an optional step is normalization, that is, scaling results by a reference set of values to ease interpretation [16]. Given the national level of our analysis, here we use a normalization set reflecting U.S. totals for each environmental and human health impact category. Health care sector totals are divided pairwise by this normalization set to arrive at the national percentages for each impact category. Normalization sets have been estimated previously for several versions of the TRACI model [17–19]. We make use of the most recent set available, calculated by Ryberg *et al.* [19] for the TRACI 2.1 model for the impact categories of global warming (in CO_2-e), acidification (in SO_2-e), criteria

air pollutants (in $PM_{2.5}$-e), eutrophication (in N-e), stratospheric ozone depletion (in CFC-11-e), and photochemical smog formation (in O_3-e) [14]. This normalization set is for year 2008 emissions. We update these to 2013 by scaling with the decrease in GHG emissions from 2008 (7,050 million tons CO_2-e) to 2013 (6,673 million tons CO_2-e), for a ratio of approximately 1.06:1. It was not possible to perform normalization for ecotoxicity potential because national estimates have not been reported with units or levels of aggregation consistent with EIOLCA outputs. For human cancer and non-cancer disease, we use estimates from Lautier *et al*. [18] that are expressed using the same reference substances for cancer and non-cancer effects as the version of TRACI implemented within the EIOLCA online tool. These are benzene equivalents for cancer and toluene equivalents for non-cancer health effects. EIOLCA outputs low and high estimates for benzene-e and toluene-e emissions. We use the average of these results. Details of how these benzene and toluene equivalents are calculated in the original TRACI model can be found in Bare [20].

Unit conversion was necessary for several impact categories where the reference substance from the TRACI results did not match the reference substance needed for subsequent analysis. For particulate matter (respiratory inorganics), the output of EIOLCA used PM_{10} as the reference substance, while our normalization set used $PM_{2.5}$. Conversion was performed using the ratio of characterization factors in the TRACI method [20]: $PM_{2.5}$ = 1.67 PM_{10}-e. For photochemical oxidation potential (smog formation), the reference substance from EIOLCA is ozone equivalents while the reference substance for the IMPACT2002+ method (used for damage assessment, below) is ethene (C_2H_4). Conversion was performed using the ratio of characterization factors in TRACI: C_2H_4 = 8.99 O_3-e. For human health cancer and non-cancer effects, the reference substances from EIOLCA are benzene (C_6H_6) and toluene (C_7H_8) equivalents while the reference substance for the IMPACT2002+ method is chloroethylene (C_2H_3Cl). Conversion was performed using the ratio of characterization factors in IMPACT2002+ for emissions to water: C_6H_6 = 0.118 C_2H_3Cl-e and C_7H_8 = 0.0127 C_2H_3Cl-e.

Results for each category are summed across U.S. expenditures to create health care sector totals, which are then compared to U.S. totals [19]. Health care-related results were translated from each emissions equivalents units of CFC-11-e (stratospheric ozone depletion, leading to skin cancer), C_2H_4-e (smog formation), $PM_{2.5}$-e (respiratory disease), and C_2H_3Cl-e (human health) to the public health metric of disability-adjusted life-years (DALYs) lost using damage assessment factors from the IMPACT2002+ model [21]. Factors used were 1.05×10^{-3} DALYs per kg CFC-11-e, 2.13×10^{-6} DALYs per kg C_2H_4-e, 7.00×10^{-4} DALYs per kg $PM_{2.5}$-e, and 2.8×10^{-6} DALYs per kg C_2H_3Cl-e emitted. Though health damages from increased ultraviolet radiation, poor ambient air quality, or toxic exposures may take many years to manifest, they are assigned to the year in which the emission took place.

Results

GHG Emissions

Fig 1 shows growth in U.S. health care GHG emissions, with an increase of more than 30% over the past decade to a total of 655 million metric tons carbon dioxide equivalents (Mt CO_2-e) in 2013, or 9.8% of the national total. If the US health care sector were itself a country, it would rank 13th in the world for GHG emissions, ahead of the entire U.K. [22]. Disaggregated results are provided in Table 1. The largest contributors to emissions by expenditure category were Hospital Care (36%), Physician and Clinical Services (12%), and Prescription Drugs (10%), not including releases of waste anesthetic gases. Expressing the results disaggregated by EIOLCA sector reveals that only 2.5% of GHG emissions were directly from the operation of health care facilities (*e.g.*, from on-site boilers), meaning that the majority of health

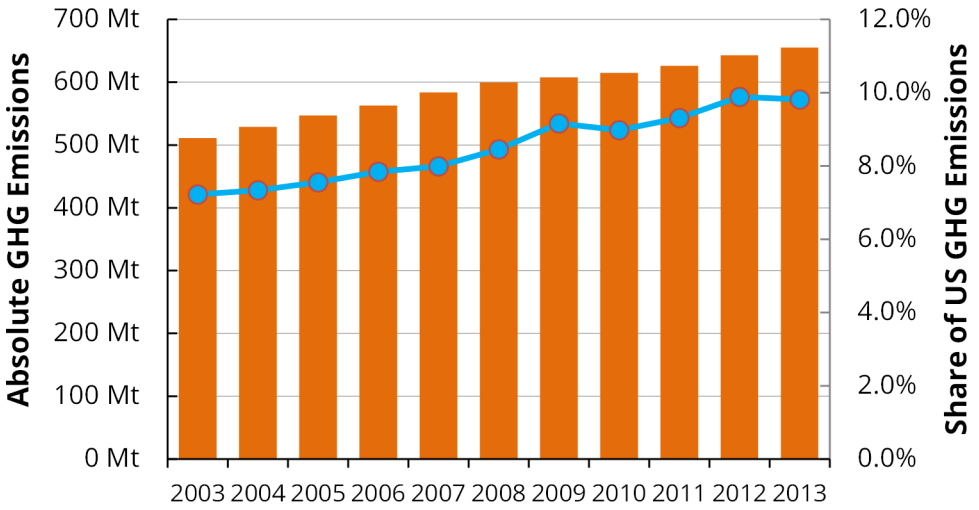

Fig 1. Time series of life cycle GHG emissions from US health care activities. Shown for 2003–2013, in absolute terms (orange bars) and as a share of U.S. national emissions (blue line). Mt = million metric tons.

doi:10.1371/journal.pone.0157014.g001

care's carbon footprint is associated with its suppliers of energy, goods, and services. Among these supplying sectors, the largest sources of GHG emissions induced by health care activities were: power generation (36%); government services (8%); non-residential commercial and health care construction (this includes "embodied carbon" of health care facilities (4%); and basic organic chemicals manufacturing (3%) (Table A in S1 File).

Table 1. Absolute health care greenhouse gas emissions (Mt CO_2-e) by National Health Expenditure category and U.S. total for 2003–2013

Expenditure category / Year	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013
Hospital Care	184	188	195	200	206	210	218	222	226	233	238
Physician and Clinical Services	57	60	62	65	65	68	69	70	72	74	77
Other Professional Services	7	8	8	8	8	8	9	9	9	10	10
Dental Services	11	12	12	12	12	12	12	12	12	12	11
Other Health, Residential, and Personal Care	20	21	22	22	23	23	24	25	25	25	26
Home Health Care	9	10	11	12	13	13	14	15	15	16	17
Nursing Care Facilities and Continuing Care Retirement Communities	35	36	37	37	38	39	39	39	40	40	41
Prescription Drugs	59	63	65	68	71	71	72	69	68	67	68
Durable Medical Equipment	12	13	14	15	16	16	16	16	17	17	18
Other Non-Durable Medical Products	11	11	12	12	13	13	13	13	14	15	15
Government Administration	13	13	14	14	13	13	13	13	14	14	15
Net Cost of Health Insurance	7	7	7	8	8	8	8	8	8	8	9
Government Public Health Activities	28	28	28	28	29	30	31	31	29	29	29
Research	12	12	13	12	12	12	12	13	12	12	11
Structures and Equipment	45	47	50	51	57	62	59	60	65	70	71
Health Care TOTAL	**511**	**529**	**547**	**563**	**584**	**600**	**608**	**615**	**626**	**643**	**655**
U.S. TOTAL[a]	7073	7208	7245	7182	7308	7096	6636	6849	6727	6502	6673
Health Care % of U.S. GHG Emissions	**7.2%**	**7.3%**	**7.6%**	**7.8%**	**8.0%**	**8.5%**	**9.2%**	**9.0%**	**9.3%**	**9.9%**	**9.8%**

[a] US national emissions are from the annual US Greenhouse Gas Emissions Inventory conducted by the USEPA.

doi:10.1371/journal.pone.0157014.t001

Several trends are evident in U.S. health care GHG emissions over the past decade. Health care spending has increased monotonically, with a slight decrease in 2011 in real dollars. As emissions are directly proportional to spending in EIOLCA models and are based on a single year (2002 in this case), emissions estimates have also increased over the decade in every spending category. (Adjustments to results based on using dynamic rather than static emission factors are discussed in the Assumptions and Uncertainty section below.) The greatest increases over the decade have been in home health care (+66%) and hospital care (+41%). At the same time, national GHG emissions as inventoried by the EPA have been trending down, largely as a result of efficiency improvements, decreased motor vehicle use, and fuel switching for production of heat and electricity.

Non-GHG Emissions

Total results for 2013 in all environmental categories are shown in Table 2, with the proportion of national totals in each category and the total disease burden, in DALYs. Through its direct and indirect emissions, U.S. health care was responsible for significant fractions of national air pollution and subsequent public health burdens, including acidification (12%), smog formation (10%) and respiratory disease from particulate matter (9%), with a lower contribution to ozone depletion and carcinogenic and non-carcinogenic air toxics (1–2%). Health damages from these five categories of pollutants are estimated at 470,000 DALYs lost due to health care activities in 2013.

Disaggregated results by health expenditure category are presented in Fig 2, with numerical details provided in S3 Table. In 2013, hospitals were the largest contributor among expenditure categories to environmental and health impacts, between 31–37% of the total. The one exception is for ozone depletion, to which prescription drug expenditures were the largest contributor at 33%, with an additional 22% from medical devices and 15% from hospital care. Health care structures and equipment contribute 17% of PM-equivalent emissions and 15% to smog

Table 2. Environmental and health effects due to health care sector direct and indirect emissions for 2013.

Effect category	Unit / Reference Substance [a]	Health Care Total	National Total	% of National	DALYs Lost [e]
GW	kg CO_2-e	6.6E+11	6.5E+12	9.8% [c]	-
AP	kg SO_2-e	3.1E+09	2.6E+10	11.7% [c]	-
PM	kg PM_{10}-e	1.0E+09	6.8E+09	8.9% [c]	435,000
EP	kg N-e	9.4E+07	6.1E+09	1.5% [c]	-
ODP	kg CFC-11-e	7.3E+05	4.5E+07	1.6% [c]	770
POP	kg O_3-e	4.0E+10	3.9E+11	10.0% [c]	9,400
ETP	kg 2,4-D-e	6.9E+07	- [b]	- [b]	-
HH canc	kg benzene-e	2.5E+08	2.6E+10	1.0% [d]	84
HH non-canc	kg toluene-e	6.9E+11	3.3E+13	2.2% [d]	25,300

Abbreviations: GW, global warming; AP, acidification potential; PM, particulate matter; EP, eutrophication potential; ODP, ozone depletion potential; POP, photochemical oxidation potential (smog formation); ETP, ecotoxicity potential; HH canc, human health cancer effects; HH non-canc, human health non-cancer effects; suffix–e, equivalents.

[a] Reference substances from the TRACI model.

[b] Ecotoxicity national totals not available as reported disaggregated for metal and non-metal emissions.

[c] Normalization from Ryberg *et al*. [19].

[d] Normalization from Lautier *et al*. [18].

[e] Calculated using endpoint characterization factors from IMPACT2002+ model; GHG, AP, EP, and ETP impacts have indirect impacts on human health but robust endpoint characterization factors do not exist.

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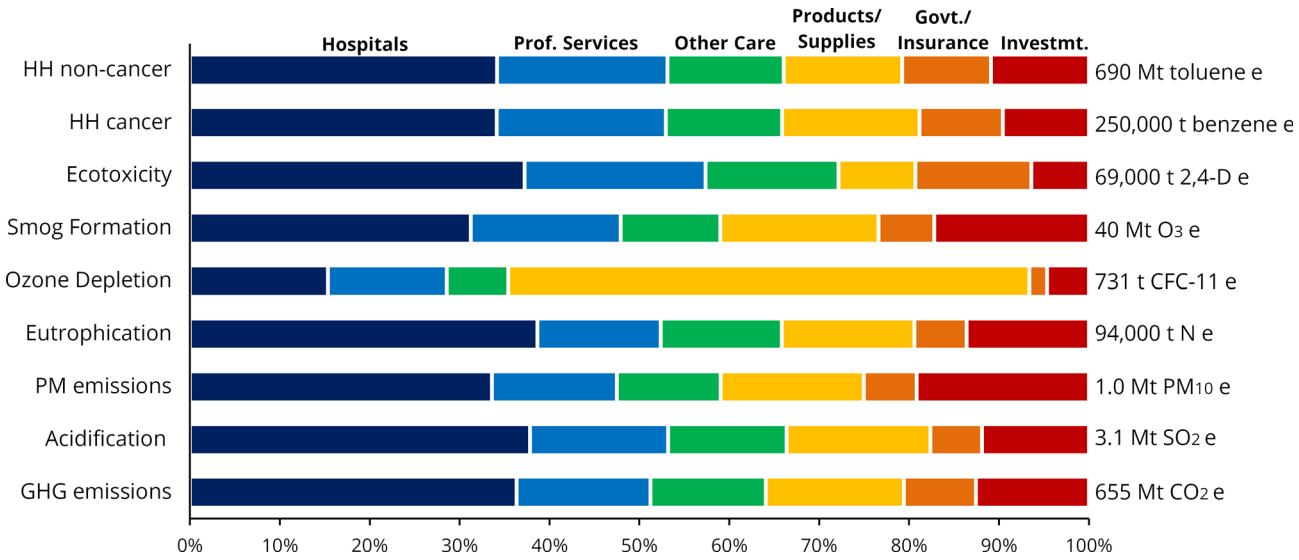

Fig 2. Environmental/health impacts of U.S. health care activities. Depicted by TRACI impact category (left vertical axis) and disaggregated by expenditure categories (colors, horizontal axis). Sector totals listed for each impact category (right vertical axis). Mt = Million metric tons, Prof. = Professional, Govt. = Government, Invstmt. = Investment.

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formation, largely from construction and manufacturing activities. Expressing the results disaggregated by EIOLCA sector (Tables A-I in S1 File) also reveals interesting patterns. 'Power generation and supply' is the largest contributing supply sector by far to acidification (44%), respiratory impacts (26%), eutrophication (21%), and smog formation (28%) impact categories, as a result of electricity use in health care facilities and their supply chains. For ozone depletion, however, the largest contributors were 'Surgical and medical instrument manufacturing' and 'Pharmaceutical preparation manufacturing' (23% each), from the use of halocarbon solvents, refrigerants, propellants, blowing agents, and other ozone depleting substances. For ecotoxicity and human health toxicity (cancer and non-cancer) the most important EIOLCA sector 'Waste management and remediation', which contributed more than 85% of the total to ecotoxicity and >50% to human health toxicity impacts.

Fig 3 shows results for all impact categories over the period 2003–2013, with numerical details provided in S4 Table. The overall increase in modeled impacts was remarkably consistent, between 27–30% across all impact categories. Ozone depletion shows the steepest slope (fastest increase) in impacts for the period 2003–2010, reflecting the large increase in ozone depletion-intensive prescription drug expenditures during that period. This increase in expenditures of 78% in real (inflation-adjusted) dollars exceeded that for all other expenditure categories. The economic slowdown during 2007–2010 can be seen in the sharp corrections for ozone depletion and respiratory effects, but after 2010 all impact categories resume consistent growth to 2013.

All of the above results are a product of life cycle emissions or effect intensities (per dollar expenditure, from the EIOLCA model) and dollars expended in each sector (from NHE accounts). The life cycle intensity values alone are presented in S5 Table for each health care-related EIOLCA sector. Per dollar of expenditure, 'Nonresidential commercial and health care structures' is the most emissions-intensive sector (on a life cycle basis, including both direct and indirect emissions) for global warming, acidification, respiratory effects, eutrophication, and smog formation, while 'Surgical and medical instrument manufacturing' is the most intensive for ozone depletion potential and 'General state and local government services' is the most

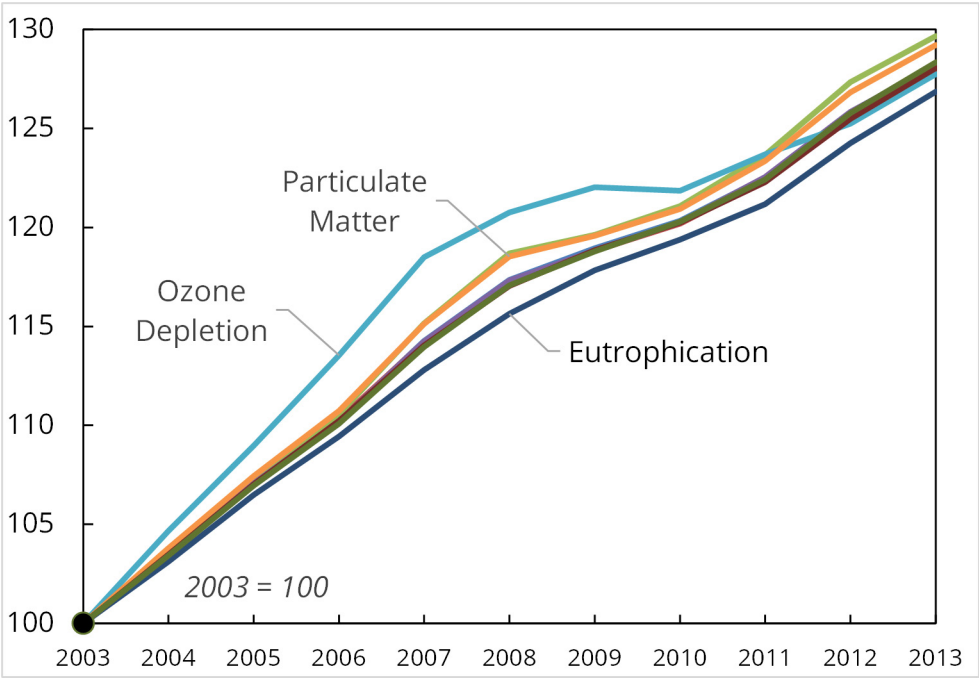

Fig 3. Time series of all life cycle impact impacts from U.S. health care activities. Shown for 2003–2013, in absolute terms.

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intensive for all toxicity impact categories, again largely due to waste management activities. Across all impact categories, 'Insurance carriers' is the least emissions-intensive EIOLCA sector related to health care, as expected for a service sector without major material or energy requirements.

Discussion

Patient Health and Public Health

Indirect health damages stemming from health sector pollution are currently unreported and largely unrecognized in health care, and it is useful to compare these results to other estimates of patient health and public health damages. The Institute of Medicine (IOM) first highlighted in 1999, *To Err is Human*: *Building a Safer Health System*, that 44,000–98,000 people die in U. S. hospitals each year as a result of preventable medical errors, at a total cost of $17–29 billion per year [23]. The IOM report highlighted that the level of preventable harm in medicine is unacceptably high, and mostly the result of faulty systems. This report sparked a national exploration into how the health care delivery system could be redesigned to innovate and improve care through formalized patient safety efforts [24] led by the U.S. Department of Health & Human Services Agency for Healthcare Research and Quality (AHRQ). With a conservative average estimate of 10 years of life lost per fatality [25], DALYs from deaths due to preventable medial errors are of the same order of magnitude as the 470,000 DALY lost due to health care-related emissions, calling for similar national attention to the need for prevention of health sector pollution.

It is important to contextualize these findings through comparison with previous work on U.S. health damages from ambient air pollution. Findings from the Institute for Health Metrics and Evaluation (IHME) for the Global Burden of Disease report, showing $PM_{2.5}$- and ozone-

related burdens of just over 1.9 million DALYs [26]. Fann *et al.* estimated $PM_{2.5}$- and ozone-related burdens of nearly 2.2 million years of life lost for 2005 (excluding transboundary emissions) [27], while Caiazzo *et al.* estimated that total combustion-related emissions in 2005 caused 210,600 premature deaths from $PM_{2.5}$ and ozone exposure [28]. While the methods used were different from the EIOLCA methods employed in the present work, suggesting caution in making direct comparisons, these past results suggest that the overall contribution of the health care sector to the national burden of disease is significant.

While some level of emissions and subsequent damages to public health are an inevitable consequence of energy use and general economic activity in our current industrial system, Americans spend more than twice as much on health care as other industrialized countries, without commensurate health benefits [29]. Just as some portion of current health care spending is excess (with a midpoint estimate of 34% in 2011 [30]) and so not necessary to maintain a high level of population health, there are opportunities to reduce health care-related emissions and indirect burdens without compromising patient care through waste reduction and efficiency improvements in the actual delivery of care. Indeed, the follow up IOM report, *Crossing the Quality Chasm*: *A New Health System for the 21st Century* [24], described six aims for quality as a systems property. These aims include avoiding injuries to patients from care that is intended to help them, as well as improving efficiency and avoiding waste. Health care pollution itself is a patient safety issue and pollution prevention ought to be included in ongoing efforts to improve health care safety and quality overall.

Assumptions and Uncertainty

Uncertainty associated with the EIOLCA model structure and construction is discussed in the model documentation and in supporting literature [12, 13]. Recall that the EIOLCA model adjoins three types of data: a matrix of economic flows (compiled by BEA), a vector of emissions for each sector (compiled from various government sources), and a matrix of characterization factors that link emissions to impacts (calculated from the EPA TRACI model). There can be uncertainty associated with values in each of these data sets. Often parameter uncertainty is unquantified because data are reported to the government directly without statistical analysis or sampling. Other sources of uncertainty include: the use of self-reported, incomplete, and/or aggregated data from industry; bias due to reporting thresholds that lead to chronic underestimation of emissions and therefore impacts (most pronounced for toxicity-related impacts); the linear structure of the model (no returns to scale); classification mismatch between BEA sectors and study sectors of interest (NHE categories in this case); and temporal mismatch between the EIOLCA model year and the study year.

Temporal considerations arise largely because of the reliance on a static economic model. Under this assumption, emissions intensities (mass of pollutants emitted per unit of economic activity) remains fixed for each economic sector, even though improvements in efficiencies and pollution control and the mix of technologies employed. (Price changes have been controlled for using the Medical Price Index as noted.) This effect is perhaps most pronounced for the power generation sector, which has experienced a dramatic decrease in the percentage of coal-fired electric generating units powering the grid over the study period, with a concomitant decrease in the carbon emissions intensity of electricity (GHG emissions per unit of electricity generated) of nearly 17% [31]. If we apply this adjustment to the share of health care GHGs from power generation (37%), this leads to a downward revision of results by ~6% to 607 Mt CO_2-e of health care GHG emissions in 2013, or 9.3% of the US national total. Considering particulate matter, according to the 2014 National Emissions Inventory (NEI), absolute emissions factors for electricity generation have decreased by 55% for primary PM_{10} and 59% for

primary $PM_{2.5}$ (including condensibles) [32], equivalent to a 57% and 61% decrease, respectively, in PM emissions factors when accounting for the increase in electricity generation from 2002–2013. Inspection of the EIOLCA results reveals that electric power generation is responsible for just 26% of total health care-related PM emissions (the percentage is 12% for the economy as a whole [32]). Thus, health damages due to PM exposure can be revised downward by approximately 15%, to 370,000 DALYs, bringing the total for all five pollutant damage categories to 405,000 DALYs. Outside of power generation, many other sectors have also experienced changes in emissions factors due to fuel switching, efficiency improvements, and improved pollution controls. For example, the truck transportation sector has experienced increases in fuel economy, improvements in engine and catalytic converter designs, and more widespread availability of ultra-low sulfur diesel fuel. It is not possible at this time to capture changing emissions trends in all of the 400+ sectors of the economy in order to adjust the results, as was done for power generation, but future versions of the EIOLCA model should provide the means to test the sensitivity of the results to changes in emission factors over time.

Other sources of uncertainty include the characterization factors that relate emissions to changes in ambient concentrations and from ambient concentrations to exposure and disease onset. Though this has been an area of intensive modeling and research recently, it is well-known that factors for human health toxicity are among the most uncertain of all impact categories, especially for toxicity stemming from metal emissions [33]. There is also uncertainty introduced by moving between model versions of TRACI and in using the separate IMPACT2002+ model to extend the TRACI results to health endpoints of DALYs lost and cases incurred, particularly for the human health cancer and non-cancer impact categories.

Future work should take advantage of upcoming model updates (to both EIOLCA and life cycle impact assessment methods) to re-run the analysis and investigate the largest contributing expenditure categories and supply sectors. Model updates may also reduce uncertainty for cancer and non-cancer impact categories including additional toxic releases in the EIOLCA inventory as well as updating characterization factors and eliminating the need for inter-model conversions. We believe, however, that the main finding is robust, namely that the health care sector is responsible for a significant proportion of emissions and public health damages in the U.S. Conversely, efforts to improve resource efficiency in health care, through energy efficiency projects or effective waste prevention and management practices, for example, will not only reap economic rewards for health care facilities but will also produce significant indirect public health benefits.

Improvement Efforts

Environmental stewardship plays a synergistic role in achieving the triple aim set out by the Institute for Healthcare Improvement and adopted by the Centers for Medicare & Medicaid Services, namely better care for individuals, better care for populations, and reducing per-capita costs [34]. Reducing waste can improve both economic and environmental performance without compromising quality of care [30]. Reducing direct and indirect emissions should be considered a key aspect of building a safer health system to improve health care quality and efficiency and reduce unintended adverse effects, both direct and indirect. Decreases in emissions that are attributable to the health care sector will have direct benefits in the U.S. and elsewhere, for example by decreasing the 3.7 million annual fatalities that result from poor ambient air quality worldwide leading to ischemic heart disease, stroke, chronic obstructive pulmonary disease, lung cancer, and acute lower respiratory infections in children [35], or the 34,000 annual cancer cases in the U.S. alone attributable to occupational and environmental exposures [36]. Economic damages from electricity generation emissions alone in the U.S. has

been estimated at in excess of $130 billion annually (dominated by health damages) [37], so controlling emissions and reducing demand for electricity could potentially save billions of dollars in health care costs. For GHGs specifically, climate change mitigation efforts have been specifically called for by the WHO and other leading health care bodies [38]. Potential health benefits include reducing the estimated 150,000 annual fatalities that occur worldwide as a result of climate change [39]. Efforts to improve the carbon footprint of health care will also have environmental and health co-benefits, as has been demonstrated for several other sectors including food and agriculture [40], urban transport and land use [41], and household energy use [42].

The World Health Organization notes the health sector, itself, can reap gains from rapid and early adoption of mitigation strategies that improve access to renewable energy, through environmentally friendly operational and building solutions [43]. In the U.S., the Healthier Hospitals Initiative (HHI) (http://healthierhospitals.org/) is a national campaign launched in 2012 to improve environmental health and sustainability in the health care sector. The HHI was organized with Health Care Without Harm, Practice Greenhealth, and The Center for Health Design, and offers tools and resources developed from the Green Guide for Healthcare. The HHI already engages 1,200+ U.S. hospitals actively seeking guidance on the transition to more sustainable operations. The American Hospitals Association also provides a Sustainability Roadmap (http://www.sustainabilityroadmap.org/.) Both offer recommendations to improve the environmental footprint of key areas that reduce both direct on-site and indirect supply chain emissions, including cleaner and more efficient energy use, water conservation, waste reduction, environmentally preferable supply chain management, safer cleaning chemicals, and healthier foods. The Coalition for Sustainable Pharmaceutical and Medical Devices is seeking to develop manufacturing standards for best practices and reporting transparency, guided by life cycle assessment (http://www.sduhealth.org.uk/areas-of-focus/carbon-hotspots/pharmaceuticals/cspm.aspx).

Clinicians play a critical, yet unaddressed role in health care resource use and represent a key opportunity for waste prevention. Seemingly small changes in how medical supplies are utilized or services delivered could have substantial benefits for resource conservation and public health when magnified over this large sector. Efforts such as the Choosing Wisely Campaign (http://www.choosingwisely.org/) offer evidence-based guidance to reduce unnecessary medical tests, treatments and procedures. A critical knowledge gap exists in the medical community regarding the indirect health consequences of wasteful, non-value added practices in all their forms, making resource conservation education and leadership crucial to improving the health system.

Conclusions

The fundamental tenet of health care practice is '*Do no harm*,' but ironically, the practice of health care itself causes significant pollution, and, consequently, indirect adverse effects on public health. We quantify life cycle emissions of the health care sector, including upstream and downstream activities, and estimate the magnitude of subsequent impacts to human health. We found this amount of disease burden, unreported and largely unrecognized in health care, is similar in magnitude to annual deaths stemming from preventable medical errors first reported in *To Err is Human* [23], which is a topic of national discourse and institutional efforts to improve health care safety. These findings underscore the need to measure, mitigate, and educate on the considerable human health and environmental impacts associated with health care practice itself. Efforts to improve the environmental performance of health care can reduce expenditures directly through waste reduction and energy savings, but also

indirectly through reducing the pollution burden on public health, and ought to be included in efforts to improve health care quality and safety.

Supporting Information

S1 File. Health care impacts by EIOLCA economic sector for 2013 (top 25 sectors).
(DOCX)

S1 Table. Mapping between National Health Expenditure categories and EIOLCA model economic sectors.
(DOCX)

S2 Table. Medical Price Index by Health Expenditure category, 1980–2013.
(DOCX)

S3 Table. Proportional contribution to GHG and non-GHG categories by National Health Expenditure category for 2013.
(DOCX)

S4 Table. Total impacts for GHG and non-GHG categories for 2003–2013.
(DOCX)

S5 Table. Relative effect intensities of health care-related EIOLCA sectors.
(DOCX)

Author Contributions

Conceived and designed the experiments: MJE. Performed the experiments: MJE. Analyzed the data: MJE. Contributed reagents/materials/analysis tools: MJE. Wrote the paper: MJE JS.

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CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani Antonelli <gabriela@sciencecom.org>
Sent: 6/28/2017 5:39:27 AM
Subject: Reminder: SCN New Science Call today, 6/28, noon eastern

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Hi All -
This is a reminder that the next SCN new science call will be held today, June 28th at noon eastern. Please note the new dial-in information below. The proposed agenda for the call is:

AGENDA:

- Welcome new board member
- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion

If you'd like to add anything to this agenda, please let me know.

***The new call-in information for Zoom teleconferencing:**
Join from PC, Mac, Linux, iOS or Android:
https://zoom.us/j/[redacted]41

Or Telephone:

Dial: +1 646 558 8656 (US Toll) or +1 408 638 0968 (US Toll)
Meeting ID: [redacted]41

International numbers available: https://zoom.us/zoomconference?[redacted]41

Thanks to all who have RSVP'd. Hope to speak to you soon,

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
@EmilySCN

From: Emily Copeland
Sent: Thursday, June 22, 2017 1:36 PM
To: 'jpmyers@ehsic.org' <jpmyers@ehsic.org>; 'phil.landrigan@mssm.edu' <phil.landrigan@mssm.edu>; 'snyderh@email.chop.edu' <snyderh@email.chop.edu>; 'CranmerJoanM@uams.edu' <CranmerJoanM@uams.edu>; 'itescua@UCMAIL.UC.EDU' <itescua@UCMAIL.UC.EDU>; 'dickjackson@ucla.edu' <dickjackson@ucla.edu>; 'tc1u@andrew.cmu.edu' <tc1u@andrew.cmu.edu>; 'deborah_cory-slechta@urmc.rochester.edu' <deborah_cory-slechta@urmc.rochester.edu>; 'BLanphear@sfu.ca' <BLanphear@sfu.ca>; 'dozonoff@bu.edu' <dozonoff@bu.edu>; 'tschettler@igc.org' <tschettler@igc.org>; 'bernard_weiss@urmc.rochester.edu' <bernard_weiss@urmc.rochester.edu>; 'carl-gustaf.bornehag@kau.se' <carl-gustaf.bornehag@kau.se>; 'RHAUSER@hohp.harvard.edu' <RHAUSER@hohp.harvard.edu>; 'sheldon.krimsky@tufts.edu' <sheldon.krimsky@tufts.edu>; 'porris@uic.edu' <porris@uic.edu>; 'barbara.demeneix@mnhn.fr' <barbara.demeneix@mnhn.fr>; 'Tracey.Woodruff@ucsf.edu' <Tracey.Woodruff@ucsf.edu>; 'jgallen@hsph.harvard.edu' <jgallen@hsph.harvard.edu>; 'michael.antoniou@kcl.ac.uk' <michael.antoniou@kcl.ac.uk>; 'blumberg@uci.edu' <blumberg@uci.edu>; 'environsc@gmail.com' <environsc@gmail.com>; 'tyrone@berkeley.edu' <tyrone@berkeley.edu>; 'pathunt@wsu.edu' <pathunt@wsu.edu>; 'dr.karp@thehappiestbaby.com' <dr.karp@thehappiestbaby.com>; 'andreas.kortenkamp@brunel.ac.uk' <andreas.kortenkamp@brunel.ac.uk>; 'gprins@uic.edu' <gprins@uic.edu>; 'rsargis@uic.edu' <rsargis@uic.edu>; 'stahlhutr@missouri.edu' <stahlhutr@missouri.edu>; 'shanna.swan@mssm.edu' <shanna.swan@mssm.edu>; 'vomsaalF@missouri.edu' <vomsaalF@missouri.edu>; 'leonardo.trasande@nyumc.org' <leonardo.trasande@nyumc.org>; 'lvandenberg@schoolph.umass.edu' <lvandenberg@schoolph.umass.edu>; 'tzoeller@bio.umass.edu' <tzoeller@bio.umass.edu>; 'kaleekreider@gmail.com' <kaleekreider@gmail.com>; 'sar.vogel@gmail.com' <sar.vogel@gmail.com>; 'Frank.vonHippel@nau.edu' <Frank.vonHippel@nau.edu>
Cc: Amy Kostant - Science Communication Network (amy@sciencecom.org) <amy@sciencecom.org>; Gabriela Silvani (gabriela@sciencecom.org) <gabriela@sciencecom.org>
Subject: SCN New Science Call next Wednesday, 6/28, noon

Hi all -
This is a reminder that this month's SCN new science call will be held next **Wednesday, June 28th at noon eastern**. If you'd like to present new or soon-to-be released science, discuss a particular topic or share key points from a recent conference, please let me know so I can include it on the agenda.

***Please note the new call-in information:**

Join from PC, Mac, Linux, iOS or Android:
https://zoom.us/j/[redacted]

Or Telephone:
Dial: +1 646 558 8656 (US Toll) or +1 408 638 0968 (US Toll)
Meeting ID: [redacted]

International numbers available: https://zoom.us/zoomconference[redacted]

Thanks in advance to those of you who let me know if you plan to join the call.

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
@EmilySCN

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 10/28/2015 5:41:54 AM
Subject: Reminder: SCN New Science call today, 10/28, noon eastern

---

Hi all -

This is a reminder that the next SCN new science call will be held today at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: 41

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org
@EmilySCN

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "lou.guillette@gmail.com" <lou.guillette@gmail.com>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 1/27/2015 8:51:18 AM
Subject: Reminder: SCN New Science Call tomorrow, 1/28, noon

This is a reminder that the next SCN new science call will be held tomorrow, January 28th at noon eastern. The proposed agenda for the call is:

AGENDA :

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion
    - European Council of Plasticizers and Intermediates (ECPI) systematic phthalate review'

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: 41

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org

From: Emily Copeland
Sent: Wednesday, January 21, 2015 10:58 AM
To: 'tc1u@andrew.cmu.edu'; 'CranmerJoanM@uams.edu'; 'deborah_cory-slechta@urmc.rochester.edu'; 'pldefur@igc.org'; 'sgilbert@innd.org'; 'lou.guillette@gmail.com'; 'tyrone@berkeley.edu'; 'heilig@sfms.org'; 'pathunt@wsu.edu'; 'dickjackson@ucla.edu'; 'dr.karp@thehappiestbaby.com'; 'phil.landrigan@mssm.edu'; 'BLanphear@sfu.ca'; 'JPMyers@ehsic.org'; 'hlnlead@pitt.edu'; 'porris@uic.edu'; 'dozonoff@bu.edu'; 'gprins@uic.edu'; 'tschettler@igc.org'; 'snyderh@email.chop.edu'; 'shanna.swan@mssm.edu'; 'vomsaalF@missouri.edu'; 'bernard_weiss@urmc.rochester.edu'; 'WoodruffT@obgyn.ucsf.edu'; 'tzoeller@bio.umass.edu'; 'shuk-mei.ho@uc.edu'; 'stahlhutr@missouri.edu'; 'blumberg@uci.edu'; 'svogel@edf.org'; 'lvandenberg@schoolph.umass.edu'; 'carl-gustaf.bornehag@kau.se'; 'RHAUSER@hohp.harvard.edu'; 'leonardo.trasande@nyu.edu'; 'kkreider@unfoundation.org'; 'itescua@UCMAIL.UC.EDU'; 'michael.antoniou@kcl.ac.uk'; 'sheldon.krimsky@tufts.edu'
Cc: Amy Kostant - Science Communication Network (amy@sciencecom.org); Gabriela Silvani (gabriela@sciencecom.org)
Subject: SCN New Science Call next Wednesday, 1/28, noon

Hi All -
This is a reminder that this month's SCN new science call will be held next **Wednesday, January 28th at noon eastern**. If you'd like to present new or soon-to-be released science, discuss a particular topic or share key points from a recent conference, please let me know so I can include it on the agenda.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: [redacted]41

Thanks in advance to those of you who let me know if you plan to join the call.

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 2/23/2016 7:08:53 AM
Subject: Reminder: SCN New Science call tomorrow, 2/24 noon eastern

---

Hi all -
This is a reminder that the next SCN new science call will be held tomorrow, February 24th at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion
    - News and Views commentary by vom Saal & Welshons in *Nature Medicine*
    - GBHs consensus statement in *Environmental Health*

If you'd like to add anything to this agenda, please let me know.

Please note the call-in information:
Dial in: 1-888-537-7715
Code: 41

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,
Emily

Emily Copeland
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202-701-8000
emily@sciencecom.org
@EmilySCN

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "lou.guillette@gmail.com" <lou.guillette@gmail.com>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 2/24/2015 8:55:06 AM
Subject: Reminder: SCN New Science call tomorrow, 2/25 noon eastern

---

Hi all -

This is a reminder that the next SCN new science call will be held tomorrow, February 25th at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: [redacted]

Hope to speak to you tomorrow,

Best,

Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "lou.guillette@gmail.com" <lou.guillette@gmail.com>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 3/24/2015 7:20:02 AM
Subject: Reminder: SCN New Science call tomorrow, 3/25 noon eastern

---

Hi all -

This is a reminder that the next SCN new science call will be held tomorrow, March 25 at noon eastern. Given recent events in the news, Ken Cook from EWG will join the call to talk about TSCA, and Maricel Maffini and Tom Neltner will join us to talk about food additives/GRAS. Thus, the agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion
    - TSCA reform Ken Cook
    - Glyphosate as a probable carcinogen
    - Food additives/obesity Tom Neltner Marcel Maffini

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715

Code: [redacted]

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "lou.guillette@gmail.com" <lou.guillette@gmail.com>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 5/11/2015 8:10:40 AM
Subject: Reminder: SCN New Science call tomorrow, 5/12 noon eastern

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Hi all -

This is a reminder that the next SCN new science call will be held tomorrow, May 12 at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: [redacted]

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,

Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org

From: Emily Copeland <emily@sciencecom.org>
To: "jpmyers@ehsic.org" <jpmyers@ehsic.org>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "tschettler@igc.org" <tschettler@igc.org>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>, "porris@uic.edu" <porris@uic.edu>, "barbara.demeneix@mnhn.fr" <barbara.demeneix@mnhn.fr>, "Tracey.Woodruff@ucsf.edu" <Tracey.Woodruff@ucsf.edu>, "jgallen@hsph.harvard.edu" <jgallen@hsph.harvard.edu>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "blumberg@uci.edu" <blumberg@uci.edu>, "environsc@gmail.com" <environsc@gmail.com>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "andreas.kortenkamp@brunel.ac.uk" <andreas.kortenkamp@brunel.ac.uk>, "gprins@uic.edu" <gprins@uic.edu>, "rsargis@uic.edu" <rsargis@uic.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "leonardo.trasande@nyumc.org" <leonardo.trasande@nyumc.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "kaleekreider@gmail.com" <kaleekreider@gmail.com>, "sar.vogel@gmail.com" <sar.vogel@gmail.com>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani Antonelli <gabriela@sciencecom.org>
Sent: 5/30/2017 7:40:53 AM
Subject: Reminder: SCN New Science call tomorrow, 5/31, noon eastern

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Hi all -
This is a reminder that the next SCN new science call will be held tomorrow, May 31st at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: 41

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,

Emily

Emily Copeland
Science Communciation Network (SCN)
202-701-8000
@EmilySCN

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "lou.guillette@gmail.com" <lou.guillette@gmail.com>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>
CC: Amy Kostant <amy@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 6/23/2015 9:23:58 AM
Subject: Reminder: SCN New Science call tomorrow, 6/24 noon eastern

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Hi all -

This is a reminder that the next SCN new science call will be held tomorrow, June 24th at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: [redacted]

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,

Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org
@EmilySCN

From: Amy Kostant <amy@sciencecom.org>
To: "jpmyers@ehsic.org" <jpmyers@ehsic.org>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "tschettler@igc.org" <tschettler@igc.org>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>, "porris@uic.edu" <porris@uic.edu>, "barbara.demeneix@mnhn.fr" <barbara.demeneix@mnhn.fr>, "Tracey.Woodruff@ucsf.edu" <Tracey.Woodruff@ucsf.edu>, "jgallen@hsph.harvard.edu" <jgallen@hsph.harvard.edu>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "blumberg@uci.edu" <blumberg@uci.edu>, "environsc@gmail.com" <environsc@gmail.com>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "andreas.kortenkamp@brunel.ac.uk" <andreas.kortenkamp@brunel.ac.uk>, "gprins@uic.edu" <gprins@uic.edu>, "rsargis@uic.edu" <rsargis@uic.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "leonardo.trasande@nyumc.org" <leonardo.trasande@nyumc.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "kaleekreider@gmail.com" <kaleekreider@gmail.com>, "sar.vogel@gmail.com" <sar.vogel@gmail.com>, "Frank.vonHippel@nau.edu" <Frank.vonHippel@nau.edu>, "drdavidmichaels@gmail.com" <drdavidmichaels@gmail.com>
CC: Emily Copeland <emily@sciencecom.org>, Gabriela Silvani Antonelli <gabriela@sciencecom.org>
Sent: 7/25/2017 4:53:25 AM
Subject: Reminder: SCN New Science call tomorrow, 7/26 noon eastern

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Hi All -

This is a reminder that the next SCN new science call will be held tomorrow, July 26th, at noon eastern.

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion

If you'd like to add anything to this agenda, please let me know.

***Please note the new call-in information:**
Join from PC, Mac, Linux, iOS or Android:
https://zoom.us/j/[redacted]

Or Telephone:
Dial: +1 646 558 8656 (US Toll) or +1 408 638 0968 (US Toll)

Meeting ID [redacted]41

International numbers available: https://zoom.us/zoomconference[redacted]41

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,
Amy

Amy Kostant
Science Communication Network (SCN)
0: 301-654-6665
C: 202-255-6665
amy@sciencecom.org

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "lou.guillette@gmail.com" <lou.guillette@gmail.com>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>
CC: Amy Kostant <amy@sciencecom.org>
Sent: 7/28/2015 8:12:53 AM
Subject: Reminder: SCN New Science call tomorrow, 7/29 noon eastern

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Hi all -

This is a reminder that the next SCN new science call will be held tomorrow, July 29th at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion
    - EPA/NIEHS Risk Assessment Workshop report

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code: 41

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,
Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org
@EmilySCN

From: Emily Copeland <emily@sciencecom.org>
To: "tc1u@andrew.cmu.edu" <tc1u@andrew.cmu.edu>, "CranmerJoanM@uams.edu" <CranmerJoanM@uams.edu>, "deborah_cory-slechta@urmc.rochester.edu" <deborah_cory-slechta@urmc.rochester.edu>, "pldefur@igc.org" <pldefur@igc.org>, "sgilbert@innd.org" <sgilbert@innd.org>, "tyrone@berkeley.edu" <tyrone@berkeley.edu>, "heilig@sfms.org" <heilig@sfms.org>, "pathunt@wsu.edu" <pathunt@wsu.edu>, "dickjackson@ucla.edu" <dickjackson@ucla.edu>, "dr.karp@thehappiestbaby.com" <dr.karp@thehappiestbaby.com>, "phil.landrigan@mssm.edu" <phil.landrigan@mssm.edu>, "BLanphear@sfu.ca" <BLanphear@sfu.ca>, "JPMyers@ehsic.org" <JPMyers@ehsic.org>, "hlnlead@pitt.edu" <hlnlead@pitt.edu>, "porris@uic.edu" <porris@uic.edu>, "dozonoff@bu.edu" <dozonoff@bu.edu>, "gprins@uic.edu" <gprins@uic.edu>, "tschettler@igc.org" <tschettler@igc.org>, "snyderh@email.chop.edu" <snyderh@email.chop.edu>, "shanna.swan@mssm.edu" <shanna.swan@mssm.edu>, "vomsaalF@missouri.edu" <vomsaalF@missouri.edu>, "bernard_weiss@urmc.rochester.edu" <bernard_weiss@urmc.rochester.edu>, "WoodruffT@obgyn.ucsf.edu" <WoodruffT@obgyn.ucsf.edu>, "tzoeller@bio.umass.edu" <tzoeller@bio.umass.edu>, "shuk-mei.ho@uc.edu" <shuk-mei.ho@uc.edu>, "stahlhutr@missouri.edu" <stahlhutr@missouri.edu>, "blumberg@uci.edu" <blumberg@uci.edu>, "svogel@edf.org" <svogel@edf.org>, "lvandenberg@schoolph.umass.edu" <lvandenberg@schoolph.umass.edu>, "carl-gustaf.bornehag@kau.se" <carl-gustaf.bornehag@kau.se>, "RHAUSER@hohp.harvard.edu" <RHAUSER@hohp.harvard.edu>, "leonardo.trasande@nyu.edu" <leonardo.trasande@nyu.edu>, "kkreider@unfoundation.org" <kkreider@unfoundation.org>, "itescua@UCMAIL.UC.EDU" <itescua@UCMAIL.UC.EDU>, "michael.antoniou@kcl.ac.uk" <michael.antoniou@kcl.ac.uk>, "sheldon.krimsky@tufts.edu" <sheldon.krimsky@tufts.edu>
CC: Amy Kostant <amy@sciencecom.org>
Sent: 9/29/2015 1:10:05 PM
Subject: Reminder: SCN New Science call tomorrow, 9/29 noon eastern

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Hi all -
This is a reminder that the next SCN new science call will be held tomorrow, September 29th at noon eastern. The proposed agenda for the call is:

AGENDA:

- Call for new science; updates on conferences; etc.
- Dear Journalist notes and media response
- Discussion
    - Endocrine Society Statement on Endocrine-disrupting Chemicals
    - Least Terns paper

If you'd like to add anything to this agenda, please let me know.

***Please note the call-in information:**
Dial in: 1-888-537-7715
Code [redacted]

Thanks to all who have RSVP'd. Hope to speak to you tomorrow,

Best,

Emily

Emily Copeland
Science Communication Network (SCN)
202-701-8000
emily@sciencecom.org
@EmilySCN

From: Amy Kostant <amy@sciencecom.org>
To: "Hunt, Pat (pathunt@vetmed.wsu.edu)" <pathunt@vetmed.wsu.edu>
Sent: 7/19/2017 7:50:06 AM
Subject: reporter interest

Hi Pat,
I think youll hear from Brian Bienkowski at EHN soon. And Valerie Brown (excellent free-lancer) has asked for the paper, but not to write on now. She said she plans to incorporate it into a larger story later.
Cheers,
Amy

Amy Kostant
Science Communication Network (SCN)
0: 301-654-6665
C: 202-255-6665
amy@sciencecom.org

From: Amy Kostant <amy@sciencecom.org>
To: "Hunt, Pat (pathunt@vetmed.wsu.edu)" <pathunt@vetmed.wsu.edu>, "'Laura Vandenberg (lvandenberg@schoolph.umass.edu)'" <lvandenberg@schoolph.umass.edu>
CC: Emily Copeland <emily@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 2/2/2015 12:13:19 PM
Subject: reporter maybe in touch

Hi All

Shia Levitt is a reporter who does occasional pieces for NPR. Shes good. Shes interested in talking with someone about endocrine disruptors and plastics from a broad, big picture perspective, who wont bog down in one particular EDC or specific health outcome. Shes already talked with folks at the Endocrine Society (including the wonderful Tom Zoeller), as well as scientists studying one particular type of EDC, and she has an interview coming up with someone from NIEHS. Shes also the mother of an almost 2 year old and a 4 year old, so like most mothers, shes wondering what she should be worried about.

She asked if I know any great speakers on this topic who are good at breaking things down in laymen's terms. She asks why people should be concerned (or if they should be) about this when there are plenty of toxic chemicals we know for sure cause harm, like smoke from diesel fuel. Shed like to know how is the type of harm we're talking about different when we're talking about EDCs vs other toxins? What is it about the science that makes this research so challenging to actually prove? (observational studies with many variables and no control groups?)

It took about a nano-second for both of you to come to mind so I hope you dont mind that Ive given Shia your short bio and email address, and told her she would enjoy talking with either or both of you. Please let me know if she gets in touch.

All best,
Amy

From: Pete Myers <jpmyers@ehsic.org>
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Sent: 11/15/2016 10:08:13 AM
Subject: Revealing the roots of "Sense about Science"

Nov 15 Seeding doubt: How self-appointed guardians of sound science tip the scales toward industry. With its funding roots deep in the corporate sector, including tobacco, oil and gas, chemicals and pharmaceuticals, the lobbying organization Sense About Science should not surprise anyone as it promotes anti-regulatory voices. The Intercept, Food & Environment Reporting Network

Nice to see the information about funding sources of the now defunct Stats and everyones favorite manufacturer of doubt, Trevor Buttersworth

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CC: Emily Copeland <emily@sciencecom.org>, Gabriela Silvani Antonelli <gabriela@sciencecom.org>
Sent: 7/30/2017 9:26:26 AM
Subject: Sad news about Lizzie Grossman

Lizzie was a terrific friend to environmental health and green chemistry.
With sadness: http://www.opb.org/news/article/elizabeth-lizzie-grossman-journalist-portland-obit/#.WX0Hw-ZXwpo.facebook

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Sent: 7/19/2017 6:50:02 AM
Subject: sad news about Lizzie Grossman

Dear All,

Im writing with sad news about our journalist friend, Lizzie Grossman. Lizzie has cancer and has entered hospice care. I know most of you have worked with her, some of you extensively, and if youd like to send a card shes at Legacy Hopewell House Hospice, 6171 S.W. Capitol Highway, Portland, OR 97239-2649.

Lizzies been a terrific friend to environmental health science, advancing awareness and understanding of the field for many years.

With sadness,

Amy

From: Amy Kostant <amy@sciencecom.org>
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CC: Emily Copeland <emily@sciencecom.org>, Gabriela Silvani <gabriela@sciencecom.org>
Sent: 2/1/2017 9:09:15 AM
Subject: Science Communication Fellows

Attach: [GCSciComFellows 2017.pdf]
Hi All,

Heres the flyer for the Science Communication Fellows program I mentioned on the call last week.

The fellowship is open to scientists from sustainable science fields, including green chemists and environmental health scientists. Participants will be postdoctoral students, university faculty and/or early career scientists in government or private industry with active research programs related to the environmental health sciences and/or green chemistry.

With warm wishes,

Amy

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Science Communication

Science Communication Fellows Program

Call for Applications

The Science Communication Fellowship program trains future scientific leaders to engage with journalists and the public about rapidly evolving research associated with green chemistry and environmental health sciences.

The Fellowship is for early-career scientists seeking to communicate effectively about complex science, without "dumbing it down", so that it may have more value and impact.

Now, more than ever, it is important that scientists help the public value science and its role in shaping our future.

The fellowship is open to scientists from sustainable science fields, including green chemists and environmental health scientists. Participants will be postdoctoral students, university faculty and/or early career scientists in government or private industry with active research programs related to the environmental health sciences and/or green chemistry.

Learn more, visit www.advancinggreenchemistry.org/collaborations/science-communication-fellows.

Details

Up to 10 Fellows will be chosen by a panel of experts through an application process.

Fellows participate in monthly video meetings where fellows present research studies and participate in mini-trainings.

Fellows present their personal research, telling the story of why it is important.

Fellows must attend a two-day, in person, all expenses paid group training at the start of the fellowship; other program work is done from home or office, by phone and the Internet.

Fellows must be in North America and fluent in English. Each receives a $2,500 stipend.

The program runs for 12 months, beginning in May 2017.

Applications Due February 28

Advancing Green Chemistry

SCN Science Communication Network

Environmental Health Sciences
www.EnvironmentalHealthNews.org

The Science Communication Fellows Program is sponsored by

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CC: Emily Copeland <emily@sciencecom.org>
Sent: 2/23/2017 12:52:21 PM
Subject: Science Communication Fellows

Hi All,

Several of you have asked that I resend this. If you know someone(s) who would make good use of the program, I hope youll recommend them and encourage them to apply.

The fellowship is open to scientists from sustainable science fields, including green chemists and environmental health scientists. Participants will be postdoctoral students, university faculty and/or early career scientists in government or private industry with active research programs related to the environmental health sciences and/or green chemistry.

For details on how to recommend or apply: http://advancinggreenchemistry.org/collaborations/science-communication-fellows/science-communication-fellowship-2017/

Thanks to those who have already made recommendations,

Amy

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Sent: 3/2/2016 5:00:57 AM
Subject: science communication

Attach: [EMB4_2016-0301 Mermaid.jpg]

Here's a completely different approach. This is a street performance artist in Miami protesting marine plastic pollution, particularly EDCs. While it took a serious effort to get her to leave character I managed to break through her stage persona and had quite a good conversation. She's very knowledgeable and had quite an elaborate show that involved a lot of one-on-one interactions with the crowd. Many props. Some of them on the edge of propriety. If there's ever another E.hormone she's a candidate in the spirit of some of the other art John has included there.

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Sent: 3/6/2017 6:25:05 AM
Subject: Scientists and politics

Mar 06 Do scientists lose credibility when they become political? A new study suggests that, contrary to common fears, the answer is no. "Scientists may have more flexibility to engage in issue advocacy without risking their standing in the public eye than they may realize." The Atlantic